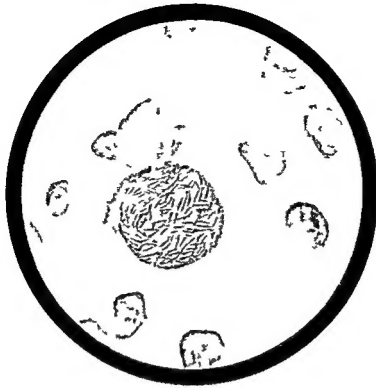




MYCOBACTERIUM LEPRAE SHOWING
INTRACELLULAR FORES



MYCOBACTERIUM LEPRAE SHOWING LARGE
GLOBUS IN THE LIVER OF A MONKEY

OXFORD MEDICAL PUBLICATIONS

A PRACTICAL TEXTBOOK OF LEPROSY

BY

R G COCHRANE

M D CbB (Glas) FRCP (Lond) DTM & H (Eng)

MEDICAL SECRETARY MISSION TO LEPROS

PRINCIPAL MISSION BY MEDICAL COLLEGE VELLORE S INDIA
HON DIRECTOR LEPROSY CAM AIGN AND HON DIRECTOR LEPROSY
RE E RCS MADRA PRESIDENCY HON PHYSICIAN IN CHARGE OF
LEPROSY DEPARTMENTS GENERAL AND STANLEY HOSPITALS MADRAS
LATELY HIEF MEDICAL OFFICE LADY WILLINGDON LEPROSY
SANATORIUM CHINGLEPU S INDIA

WITH A FOREWORD BY

GEORGE R McROBERTS

CIE MD FRCP DTM & H Lt Col IMS

GEOFFREY CUMBERLEGE

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THIS BOOK IS DEDICATED
TO MY WIFE

BUT FOR WHOSE ENCOURAGEMENT IT WOULD
NEVER HAVE BEEN ATTEMPTED AND WITH
OUT WHOSE PERSISTENCE WHEN ENERGY
WAS FLAGGING AND ENTHUSIASM WAVING
IT WOULD NOT HAVE BEEN COMPLETED

FOREWORD

By

GEORGE P McROBERT CIE MD FRCP DTM & H Lt Col I MS

Lately Professor of Medicine Madras Medical College

Physician Superintendent General Hospital Madras

Amongst qualified medical men more misconceptions exist about leprosy than about any other disease

Doctors arriving in the tropics for the first time generally believe that leprosy is a loathsome disease easily recognisable but amenable to treatment and often cured by modern therapy

On the other hand those trained in medical schools in the tropics too often take an utterly pessimistic view of the malady. They know that diagnosis is frequently difficult and treatment often futile but they have not yet grasped the importance of assessment of the type of leprosy present nor have they recognised the benign and self healing nature of some forms of the disease

The facts are that certain cases of leprosy become cured without any treatment whatsoever whilst others go from bad to worse despite our best endeavours. A fair proportion of persons infected with leprosy look perfectly normal such lesions as are present can sometimes be detected only by a trained expert and a number of such apparently healthy persons are infective

Too much success has been claimed for chaulmoogra oil and its derivatives and irresponsible optimism with regard to their value has deflected attention from the acute need for more active search for effective chemotherapeutic weapons

Dr Cochrane's experience as a teacher of post graduates and under graduate as a field worker and as a researcher in the wards and laboratory in different parts of the world amongst races of differing susceptibility enables him to write on this difficult subject with authority

His balanced and eminently sensible views on the whole problem of leprosy are worthy of the closest attention

PREFACE

— —

It has been felt for a considerable time that a practical textbook on leprosy would fill a gap between the type of book which deals largely with the theoretical aspects of the subject and the smaller pamphlets. It was after much persuasion this work was written and it is hoped that it will be of help to practitioners in the tropics, medical officers in charge of institutions as well as to the specialist.

An attempt has been made to view the subject in a comprehensive manner so that the preventive worker as well as the doctor dealing with the curative side of leprosy will find help.

It is difficult to acknowledge all the friends who have encouraged me in this task. To Colonel McRobert who for many years has been pressing me to write this book I owe a debt of gratitude for help and inspiration. To my own colleagues in the Lady Willingdon Leprosy Sanatorium I am deeply mindful of all their encouragement and of their varying and long experience which they have placed at my disposal. I have to thank Dr Herbert Ganes Medical Superintendent of the Clare Leprosy Hospital Chandluri Central Provinces for help in the chapters dealing with the surgical aspects of the subject. To Dr Donald Dow lately Medical Superintendent of the Leprosy Hospital Dichapli Hyderabad Deccan who was good enough to help in the preparation of the chapters dealing with Electrotherapy and the organisation and management of a leprosy sanatorium I would express my appreciation.

I should like particularly to express my thanks to Messrs Vale & Co of the Bangalore Photo Store for their help in preparing the prints from my negatives and for their ready assistance at all times especially during six years of war. B N Pivett Esq has given considerable technical advice in the preparation of the photo micrographs and has loaned several illustrations. Dr Alister Macleish of the Friends Ambulance Unit and Dr Norman Macpherson and Dr Edward Cault of Vellore have given useful and constructive criticism.

I am particularly grateful to my wife for helping with the preparation of the manuscript for the press and for the many hours of labour which she has put into the production.

To Dr Kate Young who generously volunteered to undertake the difficult work of preparing the index and with so little time in which to complete her task no words of mine can adequately convey my thanks and appreciation.

My publishers have shown that courtesy and appreciation of an author's difficulties which is characteristic of the Oxford University Press.

P G C

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CHAPTER I

INTRODUCTION

(a) Modern Outlook towards Leprosy

It is with some hesitation that this book has been prepared but perhaps nowhere in the world is a greater wealth of clinical material available than in the Madras Presidency where during the last ten years there has been a unique opportunity to study the practical aspects of leprosy at the Lady Willingdon Leprosy Sanatorium Chingleput and its associated clinics. In addition the author has a choice of over two thousand clinical photographs and over 700 skin sections from which to illustrate his text. This with an experience of over twenty years justifies itself the publishing of a textbook on leprosy which deals with the subject along essentially practical lines. The modern physician in the tropics is anxious to be acquainted with the disease as he is likely to see it in everyday practice and desires clear guidance with regard to the management of a case of leprosy and how to deal with its complications. This book is an attempt to meet this need. It has for the past ten or more years been impressed on us that there is no book which deals with leprosy both from the point of view of the practitioner in the tropics who inevitably must treat cases of leprosy, as well as the doctor who finds that one of his duties is the care and management of a leprosy institution. While this is essentially a practical book it is hoped that it will also be of use to the medical man who is specialising in leprosy and that it will encourage others to take up this work as a speciality for not until more medical men are willing to devote their lives to the elucidation of this in some ways the world's most baffling disease will real progress be made.

It is only within the last four years that leprosy has been considered of sufficient importance in the Madras Presidency to warrant the organisation of special leprosy departments in the teaching hospitals and it is hardly necessary to remind readers that even to day very few medical men have more than a nodding acquaintance with a disease which has puzzled mankind from the time of Hippocrates up to the present day. While this is still the case it is to be admitted that within recent years a great deal of interest in leprosy has been aroused. This has been largely due to the labours of Rogers and Muir followed later by Lowe Santra and Dhamendra in India, Mercado and Henser followed by Wade Lara and Podriguez in the Philippines, Mitsuda Hayashi and his colleagues in Japan and Lampie in the Dutch East Indies, Ryrie in Malaya and Pose in British Guiana and Robello and others in South America. The work of these pioneers is now bearing fruit and at long last leprosy is beginning to be viewed not solely as a social problem but primarily as a medical and preventive one.

It may be explained at this point that it is impossible always to state the source from which information has been gleaned for in an experience of over twenty years and with the privilege of having had contact with workers throughout the world lessons have been learnt and opinions fashioned into one's own thinking in such a way that it is very difficult to remember accurately the source of the original inspiration and

hence if there have been omissions in this direction it is hoped that they will be overlooked. I should however be lacking in courtesy if I did not express a deep sense of gratitude to all workers wherever they are who have shared with me their knowledge especially to Dr E Muir Dr Wade Dr Lowe and Dr Rynie whose friendship and guidance have widened my vision and increased my understanding.

Finally a book of this nature could not be written if it were not for the enthusiastic band of assistants. It would be impossible to name all those who have by their co-operation and loyalty given the writer the opportunity for wider and more concentrated study but one would be wanting in gratitude if mention were not made of the doctors nursing sisters compounders technicians and hospital assistants in the Lady Willingdon Leprosy Sanatorium and its associated clinics and Investigation Units who by their loyalty and devotion to duty enabled information to be collected which would otherwise have been impossible to secure.

It cannot be too strongly emphasised that in order to view leprosy aright one's whole attitude towards the disease must be altered. Everyone who commences leprosy work has to overcome to a lesser or greater extent an inborn fear of the disease. This fear has been engendered through traditional beliefs which have been passed down throughout the ages and which appear to be common to all nations and peoples where leprosy has been in existence from time immemorial. It is not only the lay public who shun those suffering from leprosy but many medical men think that this disease is outside their province and show in the presence of those suffering from leprosy quite an unwarranted fear. This fear is usually directed against the more obvious sufferer while the less obviously afflicted and frequently more infective person is allowed to mix with the public without let or hindrance. If one is to succeed in leprosy work one must adopt an attitude of friendliness even though at times stern action is inevitable. Behind all one must act the sufferer from leprosy should feel that it is realised that he is a human being with a human personality and a human soul and not a person to be shunned. Therefore such words as *leper* *clean* *untainted* should find no part in one's vocabulary because they indicate a conception which belongs to the Middle Ages.

It is interesting to note in this connection that special nouns have been used largely for diseases with a social stigma e.g. syphilitic lunatic consumptive leper. Not until the medical profession ceased to call those who were mentally afflicted lunatics and those who had tuberculosis consumptives did a fresh outlook arise. It is believed therefore that if the medical profession would refuse to refer to people with leprosy as lepers then a saner attitude towards this disease would arise and those with leprosy would no longer be shunned unnecessarily and outcasted from the community with little hope of ever being re-established even though their disease in many instances is innocuous or when they have been rendered free from infection.

It has been frequently asked if there is no need to be fearful concerning leprosy—what is the explanation of the widespread dread of the disease? In European countries this dread can be traced to the attitude adopted as the result of measures taken against leprosy in Biblical times. It should here be explained that what is described as leprosy in the Bible is I believe a generic name for a group of diseases. Some authorities doubt the existence of true leprosy in the Old Testament times. The health laws of the ancient people of Israel are among the most perfect in the world. Because the people of Israel were firmly convinced that they were a chosen people

separated unto the Lord : nothing with a permanent blemish was permitted within the camp. Therefore all mutilating all infective all unsightly conditions were placed without the camp. When the word zaraath which probably covered a large number of diseases was translated it was referred to as leprosy. Hence a sense of horror was attached to the name and a disease which does not deserve the opprobrium showered upon it became synonymous with something which conveyed a sense of fear dread and horror. It is hoped that this book will help to dispel the traditional repugnance which surrounds leprosy and will enable medical men to look upon leprosy as an ordinary disease and encourage them to take the same interest in it as they would in any of the many other diseases they are called upon to treat and prevent. To day leprosy is still viewed with a terror and dread by persons in countries where the disease is prevalent. The reason for this is most likely to be found in the fact that legends with regard to leprosy are lost in the mists of time and also in the fact that a proportion of cases small compared with the numbers who acquire the disease become mutilated and in the cases the ravages wrought by the disease have been graphically depicted in text books and novels all down the ages. This has resulted in an unreasoned prejudice against the disease which is difficult to eradicate from our minds.

(b) History of Leprosy and its Spread throughout the World

A French authority stated very truthfully that leprosy is a disease as old as the world itself.

Tradition has it that the cradle of leprosy is to be found in the upper reaches of the Nile and there are Egyptian records dating from 1350 B.C. which refer to leprosy among Negro slaves from the Sudan and Nubia. This is interesting in view of the high rates of leprosy at present in Central Africa. Some authorities suggest that leprosy spread throughout the world from Africa by way of the trade routes and possibly from East Africa across to India by sea. There is considerable evidence to indicate that leprosy spread from Africa to Egypt Asia Minor and Europe. Leprosy however has been known in China for many centuries and it is impossible now to trace its origin and therefore it can only be surmised that it was introduced from Africa via India. It is known that leprosy has been prevalent in the provinces contiguous to Tibet and Burma (e.g. Yunnan and Kinsu) for centuries and this lends some support to its introduction via ancient trade routes. It is also known that in fairly recent times leprosy spread from China to Malaya the Malayan archipelago the Pacific islands and Australia. Leprosy in Japan was almost certainly introduced from China. There are records relating to the spread of leprosy to Europe through Greece about 350 B.C. brought probably by the armies of Darius while those of Pompey carried it to Rome in 62 B.C. Galen mentions it in Germany in A.D. 180 and it had spread all over western Europe by the ninth century. This is of interest because there is a widespread belief that leprosy was introduced into Europe by the return of the Crusaders. It is said that leprosy increased during the eleventh to thirteenth centuries but the disease appeared in Europe many years before this.

The study of the rise fall and subsequent disappearance of leprosy in England is of interest to all students of leprosy. The first known leprosy hospital in England was established in Nottingham between A.D. 625 and A.D. 638 in Ireland in A.D. 809 and Wales in A.D. 950. Leprosy was apparently not prevalent in Scotland till the fourteenth century. By the latter part of the fourteenth century the disease had

begun to die out in England and had nearly disappeared by the end of the sixteenth century a Royal Commission reporting that most of the lazaar houses were by then empty. Many cases still persisted in Scotland however and the disease remained endemic in Ireland up to 1778 and in the Shetland Islands up to 1798.

The reasons for the decline of leprosy in England are of interest and various causes have been suggested. Leprosy existed in the British Isles between A.D. 638 and A.D. 1798 but it had practically died out of England by the end of the fifteenth century at a time when the diet of the people had changed from black bread and salt fish to vegetables and fresh meat. The Black Death in A.D. 1340 must have carried away many sufferers from leprosy. While the awakening of a public health conscience and better standards of living undoubtedly played an important part in the elimination of leprosy it is felt however that the strict laws of segregation whereby the sufferer was not allowed to come into contact with healthy persons (especially children) were the main factors in controlling the disease. The number of persons suffering from leprosy in England in the Middle Ages could never have reached the proportions of those with leprosy in the East for the total population of the British Isles must have been comparatively small and therefore there was a possibility that by rigid laws which in the majority of instances prevented close contact between infected and healthy persons the disease could be brought under control. Priests must have had a fair knowledge of leprosy and the easier diagnosis in the light skin made the recognition of the disease simpler and probably only a small proportion were missed. The strictness of the measures taken to prevent infection will be realised when it is stated that when a diagnosis of leprosy was made the funeral service was conducted over the infected patient the significance of which was to emphasise that he had died as far as the world was concerned. After the service the priest addressed the individual in these words: While you are diseased you will enter no house no inn no forge no mill nor in the fountain will you drink water or wash your clothes. You will not eat except by yourself or with other lepers. You will enter no church during service you will mingle with no crowd. When you speak to anyone you will stand leeward. You must always use your gloves and will touch no rope without them. You will touch no child not even your own and you will return to your cabin every night.

The epidemic of leprosy reached its height in Europe between A.D. 1000 and A.D. 1400. During this period it was stated that France had two thousand lazaar houses. There were at this time 324 lazaar houses in Great Britain 283 in England 19 in Scotland 2 in Wales and 20 in Ireland. It is felt that the greatest factor in the control of leprosy in the British Isles was through the rigid precautions which were taken reducing the chances of children becoming infected. When the possibility of infecting these highly susceptible individuals was reduced to negligible proportions the disease died out of the country. The Black Death the advancement of civilisation a public health conscience all played a part but it is believed a subsidiary part.

At the same time as leprosy was dying out in the Old World it was introduced into the New first by Columbus's soldiers and much later by the slave trade from highly endemic areas in West Africa. Areas of varying endemicity still remain in the less developed countries of Europe but owing to the reasons already stated the disease has been eliminated in the main both there and in the northern states of America except for cases imported from endemic areas.

Various authorities have estimated the number of cases of leprosy in the world and in the various countries of the Empire but any figures given can only be guess work and until much more detailed information from surveys is available there seems little purpose in burdening the mind with such detail. All that need be said is that the areas of highest incidence are found in West Africa, India, Burma, South China, Malayan archipelago and certain of the Pacific islands.

Some idea of the vagueness of the figures will be realised when authorities estimate the incidence in India as between one and five per thousand of the population. This may be so if averaged out but such a method gives no idea of the true state of the disease and in an affected presidency such as Madras there are villages with incidences of 40, 60 and as high as 120 per thousand of the population. It will be stressed later that incidences of 20 or even 30 per thousand may not be of serious import for it is not the number of cases that matter but the type and age distribution that is of serious moment. The significance of all this as far as India is concerned will be explained later. It is probably not an exaggeration to state that there may be as many as 300,000 cases of leprosy in the Madras Presidency alone. Such a statement like this must however be viewed in its right perspective for many cases belong to the benign neural type and probably give rise to no trouble to the individual nor constitute a danger to the public. Leprosy in rural areas and to a less extent in urban is essentially a sporadic disease as will be illustrated in the next chapter. One village or group of villages may have a high incidence and yet another village only a few furlongs or less away may have little or no leprosy. The importance of such an observation in connection with the epidemiology and prevention of leprosy will be apparent later.

CHAPTER II

ETIOLOGY

(a) Description of *M. Leprae*

It is now generally accepted that the causative organism of leprosy is the myco bacterium *leprae* (*M. leprae*). The bacillus was discovered by Dr Armauer Hansen and his results were first published in 1874. The bacillus belongs to the group of bacilli known as acid fast organisms and is readily stained with carbol fuchsin but is somewhat more easily decolourised with acid alcohol than the *M. tuberculosis* (see frontis piece). The *M. leprae* is seen as a uniformly stained rod straight or slightly curved varying in length from 1μ – 8μ and in breadth from 0.2μ – 0.45μ . Variations of size and shape are frequently seen and the organism may show a fragmented appearance with unstained gaps. The bacilli may be beaded and many may appear as acid fast granules. It is this pleomorphic character of the bacilli that has caused considerable discussion as to the significance of these various forms. The organisms in active cases are seen in characteristic clusters, some refer to such clusters as cigar shape bundles. In addition to the bundles of bacilli in certain advanced cases and more particularly in those races in which leprosy tends to develop more seriously and more rapidly—e.g. Mongolian and European races the organisms are seen as global masses. These are clumps of bacteria enclosed in what appears to be a capsular material. This characteristic appearance was first described by Neisser who designated these as globi. Denny (1934) described globi as disc like masses seemingly restrained by a limiting membrane within which the outermost rods are aligned somewhat concentrically. There have been many explanations of this phenomenon, some consider globi to be intracellular colonies, others to be clumps of bacilli formed within lymph spaces and another view which Denny (1934) has put forward is that they may be characteristic colonies growing within an as yet unidentified restraining membrane. The clumps of bacilli are bound together by a lipid like substance called the gloea. The cluster and under certain conditions the global masses are not only found scattered sometimes in enormous numbers throughout a slide but in the more active forms intracellular clusters and globi are also seen. From the time the bacillus of leprosy was discovered until to day there have been numerous attempts at growing it on artificial media. The present position is represented by the resolutions at the Cairo Conference (1938) on this subject which were as follows:

THE CULTIVATION OF THE LEPRAE BACILLUS

REPORT OF THE SUB COMMITTEE ON IN VITRO CULTIVATION OF *M. LEPRAE*

Majority Report

The majority of the sub committee appreciate that much work has been done on the artificial cultivation of Hansen's bacillus. The fact that results reported by various individuals or groups of workers have not in the majority of instances been duplicated by others although many attempts have been made with this end in view leads to

the opinion that the problems of the *in vitro* growth of the causative agent of leprosy have not yet been solved satisfactorily. The Committee highly commends the work of all who have laboured in this field and heartily recommends that research along this line be continued.

(Signed) H. I. HASSELTINE (Chairman)
MALCOLM H. SOULF
K. F. BIRKHAUG

Minority Report

It is the opinion of the undersigned that the causative organism of leprosy has been cultivated by Professor Kedrowsky and a few other research workers. It is urged that investigators in this field be encouraged to continue in the furrow already ploughed and on the other hand to seek for new ways but always to carry on without preconceived ideas about the strict acid fastness of the different bacterial forms occurring in leprosy material which are in my opinion only broken down stages of one and the same lower fungus.

(Signed) JOHN REYNSTIERNA

In a review (1939) on the bacteriology of leprosy the late Earl McKinley summarises the position by saying: "There does not exist to day any absolute proof that any investigator has actually succeeded in the artificial cultivation of the leprosy bacillus. There is little doubt that the *M. leprae* is a very lowly pathogen as witnessed by the fact that almost every experiment on humans has failed. It is perhaps not realised that of some 140 attempts to infect man with leprosy material there is no single record of a deliberate experimental human transmission which can be accepted as proved with the possible exception of that described by Arning (1889). There are instances where doctors have deliberately infected themselves without results. It will be shown later that the adult is relatively non-susceptible and probably only exceptionally acquires the disease."

(b) Animal Inoculation

Although the *M. leprae* was one of the earliest organisms discovered and the work of Armauer Hansen was followed by the equally brilliant work of Koch in the field of tuberculosis the fact remains that it is the one organism which has resisted culture on artificial media and every attempt to produce progressive disease in animal has up to now failed. There is little purpose served in detailing the very numerous attempts at infecting animals with leprosy during the past fifty years or more but the latest work which was stimulated by the claim of Adler (1938) that the Syrian hamster was a susceptible animal deserves mention. Following on this work and on a theory that a tuber of the *collocasia antiquorum* family was an etiological factor in leprosy Oberdoeffler and Collier claimed that they had infected monkeys by first feeding them on this tuber and then inoculating them with a nodule from an advanced case of leprosy. In an attempt to confirm these and other claims animal inoculation work has been undertaken at the King Institute (Cundy, Madras) for the past eight years. Although in one instance marked dissemination of the organism resulted from splenectomy in a monkey and inserting a nodule in the splenic stump no evidence has yet been forthcoming to indicate that we or anyone else have produced progressive disease. In the monkey occasionally is seen a rapid multiplication of the bacillus in the skin of the

abdomen contiguous to the incision. This in one instance extended to ulceration over the whole abdomen. In addition to this bacilli were found in the mesenteric glands, liver and kidney but the infection does not appear to establish itself for apparently after a varying period the bacilli disappear and 3-9 months later no trace of the inoculated nodule remains.

On feeding with *colocassia antiquorum* which must be ground up and the animal fed by a pipette the monkey after some months becomes emaciated as a result of undernourishment and an erythematous moist rash appears over the body especially on the abdomen in which acid fast bacilli are found. We are of opinion however that the bacilli are not *M. leprae* but an acid fast saprophyte for occasionally a similar organism



FIG. 1.—Monkey showing lesion on forehead after inoculation and intradermal injection of lepromin.



FIG. 2.—Same monkey showing enhanced reaction of lepromin reaction after inoculation and daily injection of lepromin.

can be found in the skin of the ear of a normal animal. Experiments in the lepromin¹ reaction have also been inconclusive. In our experience all normal monkeys are negative but on inoculation sometimes after the first at other times after repeated inoculation the lepromin reaction becomes positive. In one monkey in our series in which splenectomy was performed and a nodule fixed to the splenic stump then given daily intradermal injections of lepromin the reaction became so enhanced (see illustration) that haemorrhagic nodules were produced. When the experiment was repeated enhancement of the reaction was noted but the previous haemorrhagic nodules were not produced. In this animal one year later after a further course of intradermal injections of lepromin a lesion appeared on the forehead away from the injected area in which tubercloid changes were demonstrated. This lesion became prominent while injections were given but subsided during intervals between the injections.

¹ Lepromin is a finely ground up saline emulsion of *M. leprae* by the method of manife and will be described later.

our present state of knowledge it is difficult to assess the influence of these results. This work definitely confirms the opinion that animal may show at times semination of the *M. leprae* in their tissues but all attempts at producing progressive disease have so far failed. In this connection McKinlay (1931). As for animal experimentation we feel again that the only fair statement which can be made at the present time is that no investigator has yet succeeded in producing in experimental animal the counterpart of leprosy as it is known in man. Mention might be made

of other mycobacterial diseases in animals. A leprosy-like disease in dogs has been known for decades and those interested are referred to a critical review on this subject by Lowe (1937). So far as is known except for one doubtful instance mentioned by Marchoux there has been no record of an organism similar to *M. leprae* being found in the human tissues. There have been described within recent years an acid fast organism in many respects very similar to the *M. leprae* which infects cattle but outside the South East Indies no



Fig. 3—Plot of the organism in the tissue of monkey (Lowie, 1931, p. 400).

examples of this disease have been seen. Those interested in the subject are referred to an exhaustive review on mycobacterial disease published by Long *et al.* (1935).

While it is known that the *M. leprae* fulfils only one of Koch's postulates it can be found in the tissues of the body in every active case. Few leprologists of repute doubt that the causative organism of leprosy is the *M. leprae*. This infecting agent however seems to be very specialised and as yet is not known to live outside its normal habitat the human tissues or to infect lower animals.

In conclusion it may be stated that the position with regard to the *M. leprae* being the causative organism of leprosy has been well summarised by Black (1939). Absolute scientific proof that Hansen's bacillus is the sole etiologic agent awaits satisfactory cultural methods and susceptible experimental animals. However the constancy with which this bacillus is found in the lesions and the failure to find other agents makes its acceptance as the cause of leprosy almost universal.

CHAPTER III

EPIDEMIOLOGY

It has been pointed out by Stewart (1933) that diseases like leprosy and tuberculosis can be considered to be epidemic diseases behaving like other epidemic diseases rising to a peak and gradually declining. The curve of the epidemic cannot however be considered in terms of weeks or months but in terms of centuries. Thus viewed the epidemic of leprosy in the British Isles lasted from A.D. 600 to A.D. 1700 a period of eleven hundred years. The tuberculosis epidemic in England is declining. Some extremists consider that this is taking place in spite of increased knowledge of prevention and better methods of treatment. It is reasonable however to conclude that the more efficient the methods of control and the better the understanding of the disease the more likely are we to hasten the natural decline of epidemic diseases such as tuberculosis and leprosy.

A great deal has been written on the epidemiology of leprosy and many theories have been put forward on insufficient evidence. Because of the unreasonable dread of leprosy in the majority of communities it is unwise actively to encourage such attitudes by laying stress on epidemiological factors which are of academic importance but have little bearing in relation to the present knowledge of the disease. In studying the epidemiology of leprosy we shall endeavour to draw conclusions from facts which I believe cannot easily be refuted. All the evidence presented is largely the result of experience in South India over the last ten years. While I am aware that leprosy varies in certain important respects in other parts of India and the world I believe that the basic facts which are put forward in this chapter stand wherever the disease is highly endemic. Further all tables and figures published are taken from the records of the Silver Jubilee Clinic for the study of Child Leprosy Saidapet Madras and from the Lady Willingdon Leprosy Sanatorium Chingleput Madras Presidency.

In discussing the epidemiology of leprosy there are factors which from the evidence available appear to be of overwhelming importance. There are other factors of less importance and some influences which we consider are of little or no import liable to confuse the picture and encourage the prevailing dread and superstition with regard to the disease. These points will be discussed under the following headings: I Main Factors II Secondary Factors III Subsidiary Factors

I Main Factors

- (a) Age
- (b) Type
- (c) Contact
- (d) Race

II Secondary Factors

- (a) Diet
- (b) Sex
- (c) Migration of people

III Subsidiary Factors

- (a) Predisposing disease
- (b) Family susceptibility
- (c) Climate
- (d) Air food and water
- (e) Insect vectors
- (f) State of civilisation

I MAIN FACTORS

(a) Age of the Individual exposed

The importance of child leprosy has been emphasised by many writers in particular by Vandyke Carter McCoy Rogers Rodriguez Lampe and more recently Douilletals. It is our firm conviction that the great majority of those acquiring leprosy are infected and show manifestations of the disease before the age of 10. Rogers and Muir (1946) quoting evidence from South Russia Indian Commission (1890) Vandyke Carter McCoy and Tonkin suggest that the age in which leprosy declares itself is most likely to be in the decades 20-35. From a study of 2 000 cases in the Lady Willingdon Leprosy Sanatorium Chingleput Madras Presidency over a period of seven years the following graph and table give the age when the lesions were first noticed by the patient

TABLE I

0-4	5-9	10-14	15-19	20-24	25-34	35-49
6.3	13.4°	15.9	19.8	21.5°	16.1	7.0°

At the Silver Jubilee Clinic Sudderpet Madras the records of over 600 children were studied and Table II gives the age at which the diagnosis of leprosy was made in the various groups

TABLE II

Age Group	Number of Children with Leprosy	Percentage
0-4	90	15.2
5-9	315	42.8
10-14	332	41.0

In the graph and tables shown if we were to adopt the suggestion of Rogers and deduct 3-5 years for the latent period of leprosy the figures would afford still stronger support of our contention that leprosy is largely a child disease. Therefore from the evidence submitted we believe that the majority of persons acquire leprosy in India before the age of 20 and that many have been infected by the time they reach 10 years of age. This opinion has been confirmed by Lowe in an analysis of the survey figures of the Banlura District Bengal

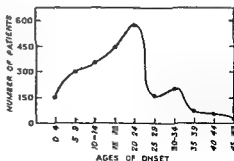


FIG. 4

when he says that the years between 3-30 are the most likely years for the signs of the disease to appear and that the maximum appears to be between 5 and 15. The importance therefore of the child in the epidemiology of leprosy cannot be too greatly stressed for we are of opinion that if children are not exposed to infection leprosy would gradually die out of a community. Thus we maintain was the main cause of the disappearance of leprosy in the British Isles. A time was reached when owing to segregation it was no longer possible for children to be infected and so the disease could not spread because we believe leprosy cannot be maintained in a community in the absence of child infection.

(b) Type of Leprosy

In the section on diagnosis details are given as to the various types of the disease here all that is necessary to say is that leprosy from the epidemiological and administrative point of view can be divided into open and closed leprosy. By open leprosy is meant that form of leprosy which is infective and from which the causal organism of leprosy can be discovered from the skin or the mucous membranes by standard methods of examination. All the evidence which has accumulated over the past seven years confirms our conviction that leprosy cannot be acquired by any other means than by contact with an open case. Considerable controversy has been raised from time to time concerning the infectivity or otherwise of closed cases of leprosy. It is of little value discussing hypothetical instances in which the evidence as to diagnosis, history and previous contact is not available or incomplete. Our experience is that as confidence is established between a community and the investigation officer the number of cases from which infection cannot be traced to an open case of leprosy becomes progressively fewer. Where it is impossible to discover the open case contact and it is chiefly among the more benign forms of leprosy in all probability the history of contact has been forgotten or possibly deliberately withheld. Table III indicates the percentage of untraced contacts in the various types of child leprosy recorded at the Silver Jubilee Children's Clinic, Saidapet.

TABLE III

Type ¹	Total	Untraced contact	Percentage
N1-Nt Major	60	36	60.0
N1-Nt Minor	225	117	51.9
N1-Ns	206	97	47.1
Incipient Lesions	38	6	18.0
Lepromatous	93	24	25.8

It will be seen that in the most serious forms of leprosy namely the incipient lesions of childhood and lepromatous leprosy over 70 per cent of the cases have been traced to an open contact. The belief in the infectivity of a closed case of leprosy is dependent on the somewhat shadowy theory that the causative organism of leprosy

¹ The classification given here and which will be described later follows in its main details the international classification of the C. I. C. O. Conference (1930).

either not the *M. leprae* or else the *M. leprae* in its life cycle passes through a yet unrecognisable form e.g. a filter passer stage or a non acid fast form but all such theories though attractive to some workers have not been supported by corresponding facts and the conclusion that we come to is that all the evidence of the past ten years indicates the soundness of the assumption that leprosy is caused by an acid fast organism known as the *M. leprae* and that the disease can only be transmitted by an open case of leprosy. On such a theory practical preventive measures can be developed and as our experience increases evidence accumulates in support of this assumption that it is only the open case or the potentially open case that need be considered in the epidemiology of leprosy.

(c) Type of Contact

If as it is maintained it is only the open case which can transmit leprosy how is the disease passed on to healthy people? It is our firm conviction that no case of leprosy can arise without previous contact with an open case and therefore the question of contact in the epidemiology of leprosy is of extreme importance. It is now generally accepted that leprosy is not a hereditary disease although the question of a family susceptibility will be discussed presently. Records from institutions in India which maintain homes for healthy children of parents with leprosy prove conclusively if evidence were needed that provided a child is separated at an early age from all contact with the disease it does not acquire leprosy. If contact is essential in the acquirement of leprosy then it is of considerable importance to determine the relative importance of the various forms of contact and for the purpose of our inquiry this can be divided into intra familial or household contact and extra familial or neighbourhood contact.

TABLE IV

Type	Total	Intrafamilial contact		Extrafamilial contact	Percentage
		Room percentage	House percentage		
Neural	20	87 42.5%	30 14.6%	89	42.9
		57.1%			
Incipient	31	24 77.4%	4 12.9%	3	9.7
		90.3%			
Lepomatous	67	43 64.3%	2 2.9%	22	32.8
		67.2%			

Table IV shows the percentage of cases of leprosy which have been traced to contact within or without the household and demonstrates the fact that not only intra familial contact but room contact is of most importance. This is to be expected because it is analogous to that other infective mycobacterial disease tuberculosis. If then children were prevented from coming into close contact within the house with open cases of leprosy this one precaution would do more towards the control of leprosy than any other single measure.

that very plausible but interesting book *Leprosy and Fish eating*. In these days of increased vitamin consciousness many of the ills to which human flesh is heir are attributed and frequently rightly so to deficiency in diet. This is always a satisfying hypothesis on which to explain any failure in treatment. Yet however satisfying or popular a hypothesis is it is incumbent on scientific workers to divest themselves of preconceived ideas and to study any theory from an unbiased view point. Diet experiments were conducted mainly among children in the Lady Willingdon Leprosy Sanatorium and Silver Jubilee Children's Clinic for over four years and no relationship could be discovered between diet and the progress of the disease or the acquirement of the more serious forms of leprosy. In the course of these dietetic experiments the following additional dietary factors were added in sufficient quantities to constitute a marked improvement in the general health of the patients—vitamins A and D in the form of Adecolin skim milk (vitamin B complex protein calcium) wheat diet (vitamin B complex protein etc). In spite of the carefully controlled experiments which were under the advisory supervision of Dr Ahroyd (Director Nutritional Enquiry Indian Research Fund Association Coonoor) it was found that there was no significant improvement in the children who were given additional dietary factors over those who had no such additions to their diet. In this connection it is interesting to note that the results of treatment in institutions where the diets are the best possible that money can obtain are no better than in those which have a relatively poor diet. While a poor diet is not advocated for in all chronic diseases it is advisable to administer as well balanced a diet as finances will allow yet it is emphasised that too much reliance should not be placed on the supposed inevitable improvement in the general condition of the patient when diets of an institution have been improved.

Why then has a dietetic factor been stressed as important in the epidemiology of the disease and what is the explanation of the observation that in areas where the diet is good frequently there is comparatively little leprosy? A possible explanation is that in those areas where the diet is adequate it is much more difficult to introduce leprosy but once leprosy has been established in a community then improvement in diet does not show a corresponding improvement in the incidence of the disease or result in a marked difference in treatment. In other words a poor diet can be likened to the careless match which starts a forest fire but once the epidemic of leprosy is established the question of diet plays little or no part in its maintenance. The non improvement with treatment is often attributed with too much facility to causes such as poor diet general health etc when the real cause needs much more careful investigation and has so far escaped us.

In this connection the latest diet theory might be mentioned. Oberdoeffler a German worker claimed that the eating of *colocassia antiquorum* was an epidemiological factor of importance in the development of serious leprosy. There is absolutely no evidence to warrant such a claim and inquiries along this line have not shown any relationship between colocassia and the incidence of severity of leprosy in South India. Lowe has stated that this theory has no sound foundation and that the consumption of colocassia as such plays no part in the causation or aggravation of leprosy.

(b) Sex

Lowe (1934) in an exhaustive review of the literature on sex incidence in leprosy stated that in most countries where leprosy is highly endemic the number of male

sufferers exceeds females by 2:1. No figures as far as is known have been given showing the sex incidence among children and adults but the following table gives the sex incidence in a population of over 3 000 examined in the Saidapet (Madras) area.

TABLE VII

Adults	Sex incidence	Children	Sex incidence
Males	51 per 1 000	Males	17.4 per 1 000
Females	32.4 per 1 000	Females	6.34 per 1 000

It will be noted that in childhood the disparity between the sexes does not appear to be of great significance and therefore it may be that the abortive lesion in female children is more frequent and thus a greater number of male children would develop serious leprosy. We have shown elsewhere that the more serious lesions increase as the massiveness of the contact increases and perhaps the male child not being so confined or reserved as the female child comes into relatively greater contact with an open case in a village or street and hence the male child may develop lesions of leprosy which more frequently tend to progress and persist into adult life. We find the following statement in an article by Hopkins and Paget (1944). That no satisfactory explanation is offered for the almost universal preponderance of males over females nor for the exception found in regard to the Negro race of whom there were admitted into the United States Marine Hospital Louisiana 25 men and 27 women. As a result of surveys in the North Arctic district and elsewhere in Madras interesting observations have been made by Sarma in which it appears that the sex incidence may be of some importance in estimating the state of the epidemic of leprosy in a community than any other single factor for there is some evidence that in areas where the female and male sex incidence is almost equal the epidemic of leprosy may be on the increase whereas in areas or communities where the disease does not appear to be spreading or where it has been established for many decades the male preponderance tends to be more marked in other words there appears to be a relationship between the duration age and intensity of the epidemic of leprosy and the sex incidence. This interesting observation which is the result of recent work done by Sarma in the Madras Presidency needs to be studied in greater detail before definite conclusions can be drawn.

(c) Migration of People

Among certain primitive communities and in villages where leprosy has not previously been in existence the disease has been introduced by persons suffering from leprosy entering the village or community. Muir and Sintra have cited such instances in the spread of leprosy in certain village communities in India and the epidemic of leprosy in the Nauru Islands was traced to the migration of Chinese some sixty or seventy years ago. This factor is of some importance not because of any increased susceptibility to infection in a so called virgin soil but in such instances the disease is frequently introduced as a result of inter marriage and hence the closest possible contact is established between the infected and non infected sources. In this way

leprosy may be introduced from many miles distant whereas contiguous villages with which there are no marriage relationships have little or no leprosy.

III SUBSIDIARY FACTORS

It is doubtful whether any of these play an appreciable role in the epidemiology of leprosy but as they are frequently considered of importance reasons for this statement must be given

(a) Predisposing Conditions and Lowered Resistance

Muir and earlier writers laid great stress on the question of lowered resistance in leprosy. The term lowered resistance has been used in a general way meaning a poor or lowered state of health. There is however little or no evidence to show that those with poor physique or concomitant diseases e.g. hookworm, dietetic deficiencies etc. have any greater tendency for leprosy to develop in the more serious forms than persons in whom no such conditions can be found. Time and again we see children and adults in a poor state of health, anaemic and with marked avitaminosis and yet with benign leprosy, and at other times we see persons apparently strong and healthy, physical examination revealing no marked ill health and yet they manifest a serious form of the disease. It is admitted that such a statement is difficult to accept but in the chapter on Pathology we hope to give further reasons why it is considered that these factors are of little importance. In fact it is very striking how comparatively frequently it is the well nourished and physically fit person e.g. policemen who acquire serious lepromatous leprosy whereas the undernourished may show benign neural lesions. All that can be stated here is that in the opinion of the writer these so called predisposing diseases and lowered state of health have been overrated as epidemiological factors. We do not advocate starvation as desirable for we consider a well nourished person means a contented person and possibly in a disease where so little is known of the exact processes of acquirement and spread spiritual and psychological factors of course scientifically not computable are the greatest assets in a return to a more satisfactory state of health. Neither can the importance of individual susceptibility be assessed. It is known that in all diseases there is great variation in individual susceptibility in persons who are equally exposed to infection and in a chronic and little understood disease such as leprosy this factor cannot be altogether ignored.

(b) Family Susceptibility

Within the last decade it has been suggested by Aycock that while leprosy is not a hereditary disease, family susceptibility to the infection may be a major factor in the epidemiology of the disease. A study of such a question is of great importance in India where one so often hears statements of the following nature from distressed patients: Doctor there is no leprosy in our family. This subject has been studied in some detail at the Silver Jubilee Children's Clinic and I only propose to present a brief summary of the findings. In an analysis of a number of families in which the exact relationship of the contact was ascertained it was found that the greatest number of children infected were from co-tenants who had no family relationship to the child. Again the percentage of children infected by the various relatives and by the co-tenants did not show such a marked difference as to suggest that the presence of family

susceptibility was a factor of outstanding importance Table VIII gives the analysis of 125 families from which these conclusions have been drawn

TABLE VIII

	Percentage infected
18 fathers have infected 27 out of 47 children	57.44
9 mothers have infected 12 out of 20 children	60.0
9 maternal uncles and aunts have infected 13 out of 28 children	46.4
16 paternal uncles and aunts have infected 21 out of 34 children	61.8
18 brothers and sisters have infected 29 out of 50 children	58.0
30 co tenants have infected 41 out of 80 children	51.2

From a detailed study of the material available the general conclusion at which we have arrived is that while it cannot be categorically denied that family susceptibility has no influence in the development of leprosy especially of the more serious types the evidence so far available indicates that this is not a major factor in the epidemiology of the disease These findings confirm the earlier work of Rodriguez (1926)

(c) Climate

This has been stressed by Powers and others and Mills (1936) suggests it is not so much the relative temperature as climatic variation which may be responsible for the spread of leprosy in other words in countries where climatic stimulation is great leprosy appears to be less common There is however little conclusive evidence that climate plays an important part in the epidemiology of leprosy

(d) Air Food and Water

There is no evidence that leprosy can be transmitted through any of these means While it is not advisable to permit open cases of leprosy to be food vendors cooks or personal servants yet the importance of the possibility of leprosy being transmitted by food or water is extremely remote and has been over emphasised For instance in India petitions not infrequently are presented to Government complaining of the fact that patients in a leprosy sanatorium may bathe in a river in which clothes are washed farther down the stream or in a tank (irrigation lake) the water of which is used for domestic purposes There are so many other much more likely sources of contact that such hypothetical reasons for infection need not be entertained as practicable or probable methods of spread Those who handle food frequently come into intimate contact with people and it is this fact not the very remote chance of food conveying the disease that constitutes the reason for prohibiting open cases having anything to do with the supply of food or water to houses

(e) Insect Vectors

Many authorities have endeavoured to incriminate insects e.g. the bed bug house fly mosquito etc. in the spread of leprosy There is very little proof however that any of these insects are the means of the spread of the disease It must be admitted that it is not impossible for leprosy to be spread by these means but in all probability they play such a small part in the transmission of leprosy that for all practical purposes

they must at best be a very minor factor in the spread of the disease. The insect which to some extent is incriminated is the *sarcoptes scabiei* for not only does this require intimate contact for infection but the parasite buries itself deep in the skin and hence if a scabietic infection is required from a person suffering from open leprosy there may be a greater possibility of contracting the disease although Rodriguez (1926) in his monograph on early leprosy in children does not admit of this¹

(f) State of Civilisation

It has been observed that in countries with an advanced state of civilisation and a well developed public health conscience leprosy does not readily spread while among aboriginal tribes e.g. Ainu of Japan and Negritos of the Philippine Islands, who have had no intimate contact with the outside world leprosy appears to be non-existent. The conditions of living among a people who have left their primitive seclusion for the greater promiscuousness of a developing civilisation may be more favourable to the spread of such diseases as leprosy. In passing it is necessary to distinguish culture from civilisation. A nation may have a high and ancient culture but according to modern standards not highly civilised on the other hand a nation may be highly civilised but poor in culture. Again undue importance cannot be attached to this factor for as members of a community advance they become more interdependent and any influence that a developing civilisation has on the requirement of leprosy can probably be attributed to the greater amount of contact which results under such conditions.

CONCLUSION

All the work in the Madras Presidency during the past ten years tends to confirm the opinion stated in this chapter that the only factors of practical importance in the epidemiology of leprosy are the age of the individual at the time of infection the closeness of contact and the massiveness of the infection. The only other influence which appears of direct importance in the epidemiology of leprosy is the race of the person exposed to infection. In other words leprosy is a contact disease and any factor which encourages overcrowding and close bodily contact is of great importance. We believe that the explanation of the acquirement of leprosy is relatively simple but because the organism has never been definitely cultivated and because there are many as yet unsolved problems this simple explanation as to how persons acquire leprosy does not always seem to be acceptable. We are of opinion that undue stress laid on factors which can at the most be only of minor importance is undesirable because attention is detracted from the overwhelmingly important epidemiological causes that of age type of disease type of contact and race and this results in effective preventive measures being delayed or not put into operation and retards the successful development of the leprosy campaign. The fact that a number of cases of leprosy may arise in European and other troops who have served in the East during the second world war does not vitiate these conclusions for these represent the few instances of persons with a high individual susceptibility and this as previously mentioned cannot be computed and may give rise to leprosy on minimal exposure.

¹ Recently Morser (194) has put forward a theory which incriminates the cockroach as a vector in leprosy but this work has received as yet no confirmation.

CHAPTER IV

PATHOLOGY

The greatest advance in leprosy during the last decade is seen in our clearer understanding of its pathological processes especially in the histopathological changes in the various forms of the disease. While Henderson enlarged on the earlier work of Muir it was not until Wade took up the study of the tuberculoid macule and drew attention to the fact that the early continental writers—e.g. Jadassohn, Lie, etc.—recognised such lesions and emphasised their importance that there was any great incentive to investigate the detailed histopathological changes in the skin. About the same time the importance of the lepromin test was increasingly realised as a result of the work of Mitsuda and Fournier followed up by Hayashi and others. All the advances have a bearing on the pathology of leprosy and we shall consider in this chapter the tissue changes in the body particularly the skin in response to invasion by *M. leprae*.

In the chapter on epidemiology we have subscribed to the view now generally held that the organism of leprosy is introduced into the body through close and generally prolonged contact with an open case of leprosy. In all probability it is through abrasions of the skin and mucous membrane of the nose that such entrance is found. Owing to the long latent period of the disease and to the inability to produce progressive lesions of leprosy in animals it is impossible to confirm the opinion that the *M. leprae* can find entrance to the body through the unbroken skin. In any case such a discussion is only of academic importance for all children and particularly those in tropical and sub tropical regions have innumerable abrasions as the result of insect bite, septic infection or scratch abrasions due to heat and other forms of irritation which constitute possible portals of entry for the organism of leprosy. In the latter part of this century Han, En, Impey and others emphasised the self healing nature of leprosy. Muir confirmed this in his observations over a period of years but it has only been emphasised more recently that of the many persons who are infected with leprosy comparatively few develop serious forms of the disease. This observation is of great importance for it robs leprosy of a great deal of its terror and is of particular significance in the prevention and prognosis of leprosy. As far back as 1926 Rodriguez indicated that early suspicious macules are changeable and evanescent and may be found a few months later to have become negative even without treatment. Little importance was attached to the significance of such a statement but in 1931 it was suggested by Cochrane that much leprosy was innocuous and because a given person showed signs of the disease it did not necessarily mean that the individual inevitably progressed and finally unless treated became an advanced case. This tendency for the body to overcome infection by *M. leprae* is well shown in Table I A which illustrates the progress of cases over the years 1937-44 at the Silver Jubilee Clinic, Saidapet, Madras among a group of children definitely diagnosed as suffering from leprosy yet receiving no treatment. When the assessment was completed in December 1944 67 children originally registered in 1937 remained in Saidapet and had been kept under observation

for seven years and a total of 249 children had been under observation over the period in question

TABLE IV

(i) Year	(ii) Total	(iii) Much improved		(iv) Improved		(v) Stationary		(vi) Worse	
		No	Percentage	No	Percentage	No	Percentage	No	Percentage
1937	67	44	65.7	7	10.4	3	4.5	14	20.9
1938	39	—	—	7	17.9	—	—	5	12.8
1939	31	19	61.3	5	16.1	3	9.7	4	12.9
1940	31	17	54.8	4	12.9	4	12.9	—	—
1941	20	13	65	4	20	9	45	9	45
1942	16	3	18.8	7	43.8	0	0	—	—
1943	5	3	60	10	200	3	60	4	80
1944	0	—	—	1	100	4	400	—	—
1937-44	49	1-4	43%	4	16.8%	43	17.7	40	16.0

Column (iii) may be further analysed as follows. In 50.8 per cent of the 14 much improved cases all lesions disappeared while in 49.2 per cent they remained as residual lesions i.e. inactive but still visible. It thus appears that amongst the children leprosy is not generally a progressive disease for in only 16 per cent has deterioration of the condition occurred, and in 50 per cent the disease has undergone spontaneous disappearance, resolution or arrest.

The above table gives striking evidence of the tendency to the spontaneous disappearance of neural lesions in childhood. Thus support is given to the contention that in the majority of the children in South India leprosy is a benign non progressive disease and that of all children who acquire the disease probably less than 50 per cent progress to the more serious forms. This subject will be dealt with in more detail in the chapter on the development of lesions. It will be asked at once what are the reasons for leprosy progressing? In the previous chapter we indicated that the four important factors in the epidemiology of the disease are age, type of disease, type of contact and race. We are of opinion also that these are the main influences which cause leprosy to become progressive. In other words the younger the person infected the more massive the infection the greater the chances are that leprosy will not only extend but will develop into the serious lepromatous form.

It is believed that a close study of the pathology of the disease especially the cellular reactions stimulated by the invasion of the M. leprae is a most fruitful line of investigation and should more surely bring us to a solution of the problem than any other avenue of research. The pathology of leprosy will now be discussed under the assumption that in the average person the M. leprae finds it very difficult to establish itself in the tissues of the body and that all tissue reactions in the presence of M. leprae are defensive in character and that the defence is more frequently successful but in a certain proportion it is unsuccessful whereas in others we see partially successful defence. In the early cases which become abortive we do not know the exact defensive mechanism but it is suggested that because M. leprae is a very lowly pathogen that under ordinary circumstances the human organism can deal with relatively small introductions of the bacilli and therefore leprosy in its clinical form never appears. Again where lesions have appeared it is possible that the M. leprae in certain instances do not multiply sufficiently to result in the stimulation of the defensive processes in

the body and that the bacilli are either rendered harmless by being confined to some lymph gland or tissue space or are dealt with by the wandering cells of the reticulo-endothelial system. It is difficult to state from the histological changes in the early macules of leprosy whether a given macule is likely to develop and progress for it is not until characteristic histological changes take place that one can estimate the probability or not of a given lesion developing into the more serious form of the disease. We cannot therefore trace the development of leprosy from the introduction of the bacilli to its more advanced stages; all that can be done is to indicate the possible method of progress and describe in more detail the characteristic histological changes in the various types of leprosy.

It is well established that there is no true incubation period in leprosy but that years may elapse between the introduction of the disease to its manifestation. Rogers and Muir give the average period as 2-3 years while infection in a child of six months has been recorded and in an adult after she left an endemic zone forty years previously. The latent period of leprosy is therefore probably a matter of year rather than months.

Elsewhere I have suggested that leprosy develops from certain basic lesions and since this publication I have no reason for altering the opinion then formed. In children then there are certain definite and characteristic histological changes in the skin and before a general discussion on the pathology of leprosy can be entered upon the changes must be described. The occurrence of an initial or primary lesion in the skin in leprosy has been accepted by Rogers and Muir (1924), Rodriguez (1936) and others and the workers have supported the ascending neural hypothesis propounded toward the end of last century. While ascending nerve infection cannot be definitely proved because as will be described later there is evidence that in certain forms the bacilli set up tissue reactions which can be traced along the nerve cords in the skin. In a disease however with such a long latent period it is considered that the first clinical evidence of leprosy does not necessarily indicate that this is an initial or primary lesion but may simply be the first external manifestation of infection with the *M. leprae*. A hypothesis will be suggested which it is claimed explains the method of development of lesions of leprosy without the necessity of assuming a primary infection at the site of inoculation. While the possibility of this is not denied it appears much more reasonable to conclude that as in tuberculosis so in leprosy infection as a rule takes place in early years and that only when conditions are favourable to the multiplication and spread of the bacilli do definite clinical lesions make their appearance. The conditions are not altogether understood but some of them have been discussed in the chapter on epidemiology.

In the development of this hypothesis it is contended as already stated that the *M. leprae* is a very lowly pathogen and that it establishes itself in the human tissue with very great difficulty. Assuming then that infection has taken place as has been indicated one of three things may happen: (a) the person may never develop leprosy and thus no indication of a previous infection is available; (b) the person may develop leprosy which shows itself in clinical lesions which may remain stationary or disappear entirely either by themselves or as a result of the development of a tissue immunity; (c) the lesions may progress and the person become an advanced case of leprosy.

The conditions under which clinical lesions develop and progress to the more serious forms of the disease are not entirely known. The wandering cells of the body

in all likelihood are probably able to deal by phagocytosis with considerable numbers of bacilli if introduced into the body but if this is impossible because of the numbers of bacilli introduced by the original inoculation or inoculation or for some other as yet unknown reason then the bacilli pass to the skin and clinical lesions make their appearance. The threshold if one may use the term above which the body cannot deal with the organism must vary with each individual. As stated in the previous chapter in addition to massive inoculation age is an important factor and apparently young persons are less able to cope with the introduction of the *M. lepra* than adults. Once clinical lesions manifest themselves the further progress of the disease seems now largely to depend on whether the body can prevent the general dissemination of the bacilli throughout the reticulo endothelial system or not. In other words it is assumed that in the development of disseminated lesions of leprosy there is a



FIG 7—Early neural case showing a concentration of round cells underneath the epidermis ($\times 700$)

cycle which must be completed. I am coming more and more to believe that unless the bacilli are disseminated via the skin no progressive lesions of leprosy can develop. If the bacilli are prevented from multiplying in the skin the disease then becomes abortive and generalised leprosy is not seen. If this cycle could be prevented either by a tissue reaction anchoring the bacilli and preventing its spread to internal organs or by some drug injected into the skin causing the skin tissue to become unfavourable to the multiplication of the bacilli then lepromatous leprosy might not be possible of development. It is known that there is a defensive mechanism anchoring the bacilli in the skin and preventing its general dissemination but where this defensive response is absent there is no means as yet known whereby it can be stimulated. The questions of diet predisposing causes etc. in preventing the dissemination of leprosy are of minor importance. It is believed that once clinical signs of the disease are manifest the prevention of more serious lesions rests with

the defensive mechanism of each individual and so far nothing is known which will improve a defensive mechanism if it is not already inherent in the individual. There is however some evidence that sub minimal infections may increase tissue immunity whereas massive infections may break down this immunity.

It is believed that the *M. leprae* is more closely related to the soil saprophytes than to the disease producing bacilli and that in its method of attack it behaves more like a parasite invading the tissue than a toxin producing organism and therefore in order to establish itself in the body certain barriers have to be broken down. It is a well known fact that pathogenic organisms have a predilection for certain tissues e.g. *B. typhosus* for the intestine the meningococcus for the meninges *M. tuberculosis* for lungs bones joints and intestines etc. The *M. leprae*, we believe shows its predilection for cutaneous tissues and if we look upon a nerve tissue as embryologically

an infolding of the primitive ectoblastic layer it can be understood why this should also be attacked. It has already been stated above that we believe that leprosy can only become a progressive disease as a result of spread from a primary focus. Where the primary focus is it is difficult to say it may be in the nerve, skin, lymph node or somewhere in the reticulo endothelial system but no clinical signs of the disease can appear except as a result of the spread of the bacilli from the primary focus and it is only when clinical signs show themselves that we can estimate whether the tissue defence which the body builds up will succeed in preventing the dissemination of leprosy. In this connection it should be noted that bacilli have been found in lymph glands of apparently normal individuals living in close contact with persons suffering from leprosy but how far the early work of Sorel and Leboeuf (1912) has been confirmed the author is unaware. It may be considered unscientific to describe the attacks of the mycobacterium leprae in military terms. As an introduction to the matter which is to follow and in an attempt to make the description clearer the defences of the body against the *M. leprae* can be likened to modern warfare where on the one hand the Russian defence was successful and the armoured thrusts of the enemy were mopped off and rendered innocuous or on the other hand where the British defence was unsuccessful as when opposed by the infiltration tactics practised by the Japanese. Bearing this analogy in mind we shall describe the defence of the tissue against the *M. leprae* in terms of successful and unsuccessful defence. We are of opinion that clinical signs of leprosy appear as a result of the activation of a primary focus the *M. leprae* multiplying showing their predilection for cutaneous and neural tissues. The first lesion frequently noted is a macule and the early macules of leprosy can be divided histologically into those lesions in which round cells are concentrated around the hair follicles and vessels in the corium and in which there is commencing invasion of nerves in the subcutaneous tissue and those lesions in which round cells and to a greater degree macrophages are scattered diffusely underneath the epidermis with no invasion of the subcutaneous nerves. It is important to recognise this difference in the histology of the early macule because the former shows potential tissue resistance and in the latter there is no such evidence. We will now discuss briefly what we believe takes place in the further development of these lesions. It is to be noted that early lesions of leprosy show histologically non specific changes the cells that are mobilised are ordinary round cells but the perivascular and perifollicular concentration of round cells indicates an early attempt on the part of the tissues to limit the infection. In individuals in whom a more marked defence is stimulated either because the bacilli are more active or because the tissues react



Fig. 8—A section from a macular (nodule) also showing commencing nerve extension in the corium ($\times 400$)

A Small nerve extension noted
B Skin tissue showing light round cell case ($\times 100$)

more strongly to the presence of a few bacilli the typical changes shown in active and effective defence are seen. At this stage it is noted that the second line of defence is mobilised and macrophages (wander cells) are conspicuous. These macrophages are arranged in typical foci or tubercles and assume the characteristic appearance of epithelioid cells. These cells have been described as cells with large pale staining oval nuclei poorly supplied with chromatin and with a broad irregular protoplasmic body which presents a homogenous appearance. The outlines of the cell body can only be made out with great difficulty in most instances. The origin of the cells is still in doubt. According to Baumgarten they arise from a proliferation of the fixed connective tissue

cells which is the result of irritation due to the presence and growth of the specific bacilli. In a publication on cytology and cell physiology edited by G. Bourne the opinion is given that epithelioid cells are transformed macrophages characterised by a central mass of fine granules stainable vitally with neutral red. The oval nucleus lies eccentrically. The following comment is made that monocytes, macrophages and epithelioid cells represent functional variations of the same cell type.

These foci because of the resemblance to changes seen in tuberculosis are called tubercloid foci and consist of a peripheral concentration of round cells with well marked epithelioid cells in the centre. The foci are seen mainly around hair follicles sweat glands and the vessels of the corium. In marked cases the epithelioid cells coalesce and form giant cells the majority of which are of the Langhans type. The result of this process is that the bacilli are anchored in the tissue and the spread of the infection prevented. When the infective process has been dealt with the cellular reaction disappears and the lesions retrograde clinically and become healed. This accounts for the spontaneous



FIG. 9.—Section from an early tubercloid lesion showing a tubercloid focus in the corium ($\times 200$).

- A Epithelioid cells
- B Blood vessel
- C Subcutaneous fat

disappearance of lesions in tubercloid leprosy and it may be noted that the more active the tissue reaction the more rapid is the resolution of the lesions. In other words to use the military analogy the spearhead of attack of the bacilli is cut off at the skin rendered innocuous and the attack aborted. This tissue anchoring process is well illustrated by the fact that the granuloma extends up to the epidermis without any separation or free space between the epidermis and tubercloid foci in the corium. It will be noted that we refer to the characteristic cell of the tubercloid foci as epithelioid following the practice of Wade rather than the term endothelioid as used by Mackay Muende and others. Further the phrase tissue immunity is used rather than such words as allergy or tissue sensitisation. Both these terms are possible of several interpretations and the general picture of a tissue anchoring process resulting in an effective defence on the part of the body seems to be a clearer conception of the pathology of this type of leprosy. If this particular reaction of the

tissues is a form of tissue immunity then all the lesions of the body should show a similar reaction in the presence of *M. leprae*. We believe that where this resistance is present whatever tissue the *M. leprae* attempt to invade tuberculoid foci are set up anchoring the invading organism and therefore the nerves in the corium show the same reaction. Hence it is difficult to say whether the infection in the nerves is an ascending infection from the skin or whether the same reaction is produced in the nerves by the simultaneous dissemination of the bacilli to the skin and the nerves from an internal focus. If this reaction is seen wherever bacilli are found why have not the histological changes of a tuberculoid nature been described in the internal organs? The reason for this we believe is that *M. leprae* can only invade pathologically the whole reticulo-endothelial system by first passing from an internal focus to the skin and then becoming disseminated throughout the whole body through lymph and blood channels. In tuberculoid leprosy the bacilli are walled off in the subcutaneous tissues and there is no possibility of their widespread dissemination. It is true that the bacilli may pass to the lymph nodes and other glands for Loe has described tuberculoid changes in lymph nodes and we have recorded a definite case of tuberculoid reaction in the lachrymal gland.

A definition of tissue immunity might clarify our minds at this point. By tissue immunity we mean the ability of the skin to anchor the bacilli and prevent their dissemination throughout the body.

It will naturally be asked whether the bacilli can be found in this type of lesion. Usually the lesions are negative to ordinary methods of examination but in the lesions showing gross tissue immunity bacilli are frequently found sometimes in large numbers but they are not usually demonstrable in the skin for more than three months the violence of the tissue reaction we assume in some way affects their nutrition and they disappear from the tissue.

It is because of the paucity of the bacilli in neural leprosy that theories as to the possibility of different forms—non acid fast—filter passing, etc.—or different strains of the organism have been conjectured. Of this there is not one iota of proof and to our mind the more reasonable explanation is that the tissues set up such an active defence that the bacilli are unable except in the more active cases to multiply sufficiently for them to be detected by ordinary means in the tissues. This form of tissue reaction may be styled effective and active cellular defence. As has been pointed out the *M. leprae* not only has a predilection for cutaneous tissue but also for neural tissue and as will be described there is a type of the disease—neural anaesthetic—in which the main if not the full brunt of the attack falls on one or more of the larger peripheral nerves—ulnar or peroneal. In this type another reaction is seen which from

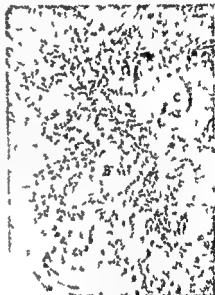


Fig. 10. Section from a tuberculoid lesion showing mode of infection. Note granuloma at its periphery (H&E).

1. 1. term
2. 2. 1 to 1 cell
C. C. nt cell

the point of view of the prevention of dissemination of the organism is just as effective a defence but results frequently in gross mutilation. The reaction is one of fibrosis and we assume that in consequence of the activation of a primary focus the bacilli pass to one or more of the larger nerves and in consequence a violent tissue reaction sets in which is not seen in the form of tuberculoid change but in an interstitial fibrosis of the nerve resulting in destruction of the bacilli with inevitable strangulation of the nerve by such a process. As stated this fibrous tissue reaction is seen particularly in the neuroanaesthetic case and to some extent in neural lesions which show macules in the skin (simple macular lesions).



FIG. 11.—Section from a major tuberculoïd lesion showing gross tissue immunity.

Note.—No free tubercle bacilli in epidermal zone and large giant cells immediately beneath the epidermis ($\times 100$).

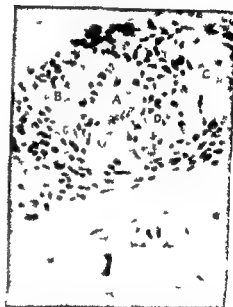


FIG. 12.—Section illustrating nerve invasion.

- A Small giant cell
 - B Epithelioid cell
 - C Low nerve bundle uninvaded
 - D Middle nerve bundle partially invaded
- ($\times 400$)

In tuberculoïd leprosy the nerves are also involved but in this instance it is not a fibrous tissue reaction which is first seen but a more active cellular type of reaction which not infrequently results in caseation and nerve abscess formation. The recognition of these two varieties is particularly important because in the fibrotic type operation is useless but in the active cellular type operation is the treatment of choice. As will be pointed out in a later chapter the decision to operate depends entirely on the nature of the tissue reaction.

We now turn to the question of the method by which the body deals with *M. lepræ* in those cases where an effective defence is not possible or development. Again we believe that the earliest lesion is a macule but in this case instead of the round cells being concentrated around hair follicles and sweat glands they are diffusely distributed

under the epidermis and in addition in an early stage wander cells or macrophages are more conspicuous. The other difference in the histological appearance is that in this instance the nerves in the corium show little or no invasion whereas in the macule which shows commencing effective tissue defence the nerves in the corium show infiltration with round cells. If as we believe the more serious form of leprosy develops from these lesions which show such vague and non-specific histology what is the process of development? We believe that the process is essentially the same.

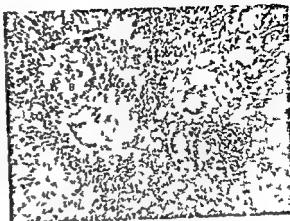


FIG. 13.—A section of skin tissue showing cellular infiltration and structural changes, likely a macule.

As a result of the multiplication of the bacilli they pass from the primary focus to the skin but in this instance the tissues are unable to anchor the bacilli and macrophages are mobilized in order to deal with the infection but instead of these cells developing into epithelioid cells and giant cells resulting in a tissue anchoring process and so preventing the dissemination of the bacilli, they become actively phagocytic and commence to ingest the bacilli. The activity of these lesions is noticed histologically by the fact that instead of



FIG. 14.—Section from minor tuberculo leprosy showing cellular infiltration and structural changes, likely a macule.

the infiltration extending up to the epidermis there is a characteristic clear zone between the epidermis and the granulomatous infiltration. These actively phagocytic macrophages are named lepra cells hence this type of leprosy in which the cells are the predominant feature is called lepromatous leprosy. In the lesions of lepromatous leprosy there is no evidence of any ability on the part of the skin tissue to cope with the bacilli the multiplication of which in the skin results in an extensive dissemination of the organism throughout the systemic system. This is first seen in the extension of the disease and fresh lesions appear in the skin all of which have the same essential histology. This process entails the spread of the bacilli from the skin to the lymphatics and to the blood stream and thus the whole of the reticulo-endothelial system become so extensively invaded that bacilli can frequently be found in the spleen, liver and even in the bone marrow. Once the organism has broken down the initial barrier

This conception of the pathology of the disease is based on the assumption that the *M. leprae* is a parasite of the reticulo endothelial system and that wherever possible the body puts up a tissue defence preventing it from infiltrating throughout that system but where this is impossible the only alternative is for the macrophages to deal with the ever multiplying bacilli by phagocytosis which results in a vicious cycle from primary lesion to skin from skin to lymph nodes spleen liver bone marrow. This type of ineffective defence has been previously likened to the Japanese tactics early in 1942 and therefore to use the graphic terms of modern times as a result of the infiltrating process the reticulo endothelial system becomes occupied territory and as yet no way has been discovered to dispossess the body of this enemy invader¹

The only organs that are grossly affected in advanced leprosy are the naso pharynx trachea the testicle and the eyes. In all these cases the pathology is not a specific

one and can be explained entirely along non specific lines. Owing to the presence of innumerable bacilli in the mouth nose and throat ulceration and secondary fibrosis is liable to result producing marked deformity and in certain cases marked ulceration and stenosis of the larynx. The presence of bacilli in the testicle gives rise to constant irritation which also ends in an interstitial fibrosis gradually destroying the organ. Bacilli invade through the blood stream and by direct spread via the naso lachrymal sac the whole choroidal coat of the eye and thus advanced cases are liable to develop an iritis iridocyclitis or even a panophthalmitis.



FIG. 19.—Section from nodular leprosy showing clearly defined in deeper part of corium around which there is quite marked granulomatous tissue almost all cells of which are lepra cells.

The reason for bacilli disappearing in the skin can only be explained we believe by the fact that as a result of their constant presence in enormous numbers the nutrition to the skin is in some way affected and they can no longer get the sustenance necessary to maintain their growth and multiplication. Some support is given to this statement because it is a well known fact that starvation or any process which causes emaciation e.g. arsenic poisoning may result in the bacilli disappearing from the skin only to return

The following quotation from the writings of Wiley D. Forbus is relevant at this point.

Macrophages which survive are not always immediately able to destroy or otherwise dispose of the ingested organism. Because of this one of the most phenomenal and significant forms of host-parasite relationship, genuine intracellular parasitism may arise. Through this at least temporary but intimate relationship between the macrophage and the organism a basically useful and distinctly protective mechanism sometimes becomes a major liability to the body as a whole. This liability arises chiefly from the wandering habits of the macrophages. The phagocytic process takes place in the injured area, but the digestive activity of the macrophages only begins there. These cells make their way rapidly into the blood capillaries and especially into the lymphatics, by the latter they are carried to the regional lymph nodes in the course of which they complete their digestive activities or they die. No doubt many of them pass on into the general circulation and there disintegrate. Thus phagocytosis becomes actually harmful since it has developed into a means of widespread dissemination of harmful agents.

when the skin recovers its normal texture. A careful and detailed research of the biochemistry of the skin might throw important light on the elements in the skin needed to nourish the bacillus and lead to more successful attempts at growth or to a line of therapeutics which would prevent the bacilli from multiplying in the skin. For if this could be done we believe that the vicious circle which results in the dissemination of the bacilli from primary focus to the skin and from the skin to the reticulo-endothelial system would be broken and the disease overcome. In the light of these remarks it can be well understood that it is possible that a well nourished skin is a better pabulum in which the bacilli can develop and multiply than an undernourished one. Therefore we believe we have a pathological basis for our contention that the place of predisposing causes in the epidemiology of leprosy has been exaggerated out of all proportion to its importance.

Before briefly discussing the nature of the tissue immunity in resistant leprosy, it might be well to consider the pathology of leprosy reaction. This condition only occurs in lepromatous leprosy and is manifested by a rapid multiplication of the bacilli and dissemination throughout the reticulo-endothelial system. Its nature has been discussed by many writers and is not altogether understood; it is not in the nature of tissue immunity because there is no attempt on the part of the cell to anchor the bacilli and it is difficult to explain on the hypothesis of a toxic condition, although cachexia after prolonged reaction does



FIG. 0.—A good example of generalized leprosy (moderately advanced) from the a.)

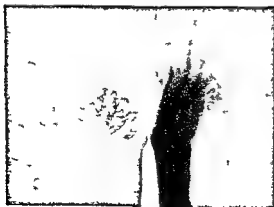


FIG. 1.—A very late stage of leprosy showing extensive ulceration of the tissue and necrosis of the hand.

set in. Rather do we feel that it is due to the stimulation of the reticulo-endothelial system causing an intense mobilisation of macrophages in their attempt to dispose of the bacilli in the lymph glands, liver and spleen. The violence of this defensive mechanism throws the body out of balance and sets up febrile attacks which may continue for months or so long as the state of acute activity exists. Sometimes this multiplication of bacilli results in such a mobilisation of cells that lymphocytes are if it may be said brought in to assist the defensive mechanism and actual pus cells are seen and the whole skin is a mass

of suppurating lesions. In other words lepra reaction is a form of defence but ineffective defence for the body cannot cope with the multiplication of the invading hosts of bacilli and deals with them in the only way possible by transferring as many bacilli as possible to the bone marrow liver spleen and throughout the reticulo-endothelial system.

While this description of the pathology of leprosy covers the majority of the instances there are groups of cases not wholly understood but which are of great importance in the question of diagnosis and prognosis. In this form an attempt at tissue immunity is seen and epithelioid and giant cells sometimes of great size are formed. The great activity of these lesions however is seen by the fact that there is no anchoring



FIG. 2.—Intermediate or border line lesion showing tuberculoid type of tissue reaction with free sub-epidermal zone ($\times 200$)



FIG. 3.—Free sub-epidermal zone at A. Enlarged capillaries B. Giant cell C ($\times 200$)

of the processes in the skin but a characteristic free and very vascular zone appears underneath the epidermis. In this reaction tissue immunity appears to commence but has a tendency to break down for there is a great accumulation of macrophages many of which are identical with the lepra cells already described. These lepra cells have a tendency to become vacuolated and the true foamy cell is not infrequently seen. Whether the bacilli are found in the systemic system is not known but this incomplete or partial immunity frequently results in the disappearance of lesions sometimes owing to gross nerve change with marked deformity at other times with little or no deformity. Whereas in tuberculoid leprosy tissue immunity is seldom if ever broken down in this type owing to the fact that there is only partial tissue immunity there is a greater tendency for the balance between bacilli and the tissue to be upset and then definite dissemination of the bacilli takes place and the patient passes into lepromatous leprosy. This partial immunity results in the development of a lesion with histological

characteristics of tuberculoid leprosy as seen by the formation of giant cells and epithelioid cells with gross nerve involvement. Side by side with these changes are seen changes characteristic of lepromatous leprosy—lepra cells, sometimes foamy cells with a free and very vascular sub-epidermal zone. It is in this type of leprosy that gross ulceration of the skin may take place and frequently to an alarming extent but the ulceration usually clears up in a few months often leaving marked scarring and deformity.

In this group of cases the tissue reactions are very varied and it is almost impossible to give an adequate picture of these changes. Suffice it to say that certain tissue reactions are akin to lepromatous leprosy, others to tuberculoid leprosy, whereas still



FIG. 4.—Typical leprosy tissue reaction showing foamy cells and giant cells (x 400).

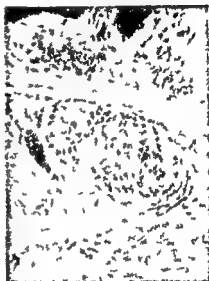


FIG. 5.—Typical leprosy tissue reaction showing nests of epithelioid cells (x 400).

others very closely resemble the sarcoids. In these varieties it is as if the defences of the body are thrown into complete confusion and every variety of wandering cell is mobilized to deal with the invader. This form of defence can only be described as partially effective because there is some evidence as seen in the occasional development of iritis in these cases that the bacilli become disseminated and it is such cases that may pass on into the lepromatous stage. Until the pathology of the intermediate or border line cases is better understood the significance of the tissue changes cannot be accurately appraised.

In the light of these statements and the relatively extremely serious outlook in lepromatous leprosy, how is one to explain the fact that a certain proportion of lepromatous cases have been known to overcome the disease as a result of intensive treatment? The method of dealing with the organism in the earlier cases and the reaction for its disappearing from the skin is little understood. It is felt that modern treatment

especially intradermal injections sets up a condition in the skin which may result in either a temporary or a permanent state in which the bacilli cannot multiply in the skin and hence the pathway for dissemination through the body is cut and as stated previously unless multiplication takes place *via the cutaneous tissue the M. leprae* appear to find it difficult if not impossible to become firmly established and remain latent in the reticulo endothelial system without showing clinical manifestation of the disease. Unsatisfactory though this explanation is there seems no other way to explain

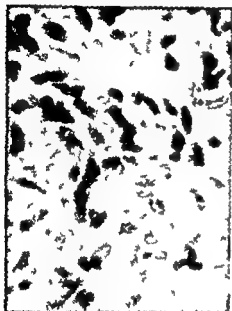


FIG 16.—In adjacent field several to my cells (b) were at cover slip ($\times 400$)



FIG 17.—High power photomicrograph of giant cell in deeper part of corium ($\times 400$)

the not infrequent recovery from the early lepromatous leprosy for there is no evidence in such cases of any immunity for should this armistice if one may term it between the cutaneous tissues and the bacilli break down the whole process of skin invasion and general dissemination of the bacillus is seen again and no immunity either tissue or humoral seems to have resulted from any previous temporary disappearance of outward signs. From this description of the pathology of leprosy it can be understood why so much stress is laid on changes in the corium of the skin. For it appears to be the only place where an effective attack on the bacilli can take place so as to stop the spread of the disease and prevent the vicious circle of re infection.

CHAPTER V

DIAGNOSTIC AND OTHER SEROLOGICAL TESTS

It will be readily understood that the question of immunity in leprosy is an extremely difficult and complicated one. For the past twenty years or more attempts have been made to discover a specific immunity test. If the conception of the pathology of leprosy described in the previous chapter is correct, then it will be readily seen why a specific serum or complement fixation test is unlikely to be discovered. The more serious lepromatous type of leprosy we believe is a parasitization of the reticulo endothelial system and this can only be accomplished as the bacilli pass from the primary focus to the skin and then throughout the reticulo endothelial system. This invasion of the reticulo endothelial system occurs only in those persons whose tissues are unable to anchor the organism and prevent its spread through the subcutaneous tissue. Hence any resistance the body is able to put up is a very specialized type of tissue resistance and is in no sense a humoral one. As there is no specific serological test of any value in the diagnosis of leprosy, it is only necessary briefly to refer to the tests which from time to time have been advocated or tried in leprosy. Over twenty years ago Taylor and Malone (1925) described a complement fixation test using as an antigen a saline suspension of tubercle bacilli. The modern counterpart of this test is that of Witebsky, Klingenstein and Kulin and Low and Crevel (1939) whom this test came to the conclusion that it was little help in the diagnosis of leprosy and was only positive in those cases which were strongly positive bacteriologically, particularly in lepromatous leprosy.

Wade (1926) made a study of the serum of patients by means of certain non specific tests, chiefly the nitric acid reaction of Bruch. This consists of a determination of excess globulin precipitate formed by nitric acid in dilute serum. Wade showed that there was some relationship between the advancement of the disease and the positiveness of the test. The test however is a non specific one and changes in the serum globulin and serum albumen ratio are noticed in other diseases e.g. kala azar. In connection with the latter disease Napier a formol gel test has been tried in leprosy and McKenzie (1933) noticed that it tended to be high not only in advanced lepromatous (nodular) leprosy but also in the presence of chronic bone leprosy.

Sedimentation Index Test

This test depends on the rate at which erythrocyte sediment when citrated blood is placed in graduated pipette. It is however not a specific test but is an index of the debility of a patient. Normally the erythrocyte sedimentation index should be below 10 but any condition which results in the breaking down of effete and dying cells increases the metabolic rate of an individual will show a rise in the sedimentation rate. This test has been largely used in rheumatic fever, tuberculous rheumatoid arthritis and many other conditions in order to gauge the progress and the activity of the particular disease. There are very few workers to day who rely on the sedimentation index test alone. For instance the tuberculo specialist uses the sedimentation

index test in conjunction with the X ray bacteriological examination of the sputum and the Arneht count giving due emphasis to each factor before endeavouring to give an opinion. Similarly we believe the sedimentation index test in leprosy cannot be used alone but must be correlated with the clinical and bacteriological findings. The greater the experience of the leprologist the less he will rely on such non specific tests as the sedimentation test. While it may be of some help in indicating the activity of the disease it is affected by too many extraneous factors to be of any real value. The implication of this statement will be discussed in the chapter on Control and Treatment.

Any standard method may be used and those interested are advised to consult larger works but the simplest technique is that advocated by Muir and is carried out as follows

0.3 cc of 5 per cent solution of sodium citrate in distilled water is drawn into an all glass 2 cc syringe. 1-2 cc of blood is drawn from the patient's vein into the same syringe and a small quantity of air having been taken into the syringe barrel the blood and citrate solution is thoroughly mixed by reversing the syringe several times and the mixture is transferred into a clean test tube. Sedimentation is carried out in 300 mm pipettes graduated from above downwards from 0 to 100. The capacity of the pipette when filled up to zero is approximately 1 cc but a variation of 0.05 cc is allowed as such a variation makes no appreciable difference in the results. Then the pipettes are placed in a rack with their points inserted in small holes bored in rubber corks.

One of these pipettes is taken from the rack and its point is inserted in one of the test tubes suction being applied by mouth at the other end of the test tube the blood citrate mixture is drawn up into the pipette to the zero mark.

The pipette is then replaced in the rack the point again being inserted in the rubber cork which prevents the mixture escaping. The rack with the pipettes should then be placed in an incubator at 37°C if this is available. If this is not available incubation at room temperature is sufficiently accurate.

The top level of the erythrocytes is read off after 1½ hours and again after 2½ hours and the average of these two readings is taken as the sedimentation index (S.I.). Thus if the level of the top of the blood cells falls to 10 after 1½ hours and to 20 after 2½ hours the S.I. will be the average of 10 and 20 i.e. 15.

Wassermann Reaction

Lloyd Mitra and Muir (1927) concluded that a positive Wassermann is due to syphilis in a great majority of cases and Pineda (1926) contended that the Wassermann reaction is negative in uncomplicated cases. Doubt has since been cast on these statements and recent work on the subject at the Lady Willingdon Leprosy Sanatorium Chingleput has tended to confirm these doubts. The work was undertaken according to the following premise. If the earlier statements that a positive Wassermann reaction is due to syphilis complicating the picture and not due to leprosy then all patients with leprosy should show improvement in the Wassermann reaction on treating them for syphilis. Ten cases were therefore chosen and treated for syphilis. The drug used was that advocated by Muir Hg 33. This is an organic compound of mercury which dissolves in hydnocarpus (chaulmoogra oil). Five of the cases were treated with Hg 33 and five left as controls. In the control group two cases both with strongly positive Wassermann and Kahn reactions became negative and in the treatment group none. The negative result corresponded with marked improvement in the clinical and bacteriological condition and suggests that leprosy was the factor which caused

the positive reaction. A further analysis of the patients with positive serum reactions¹ was undertaken and Table V gives the percentage of positive reactions in the various types in the Lady Willingdon Leprosy Sanatorium.

TABLE V

Type	Kahn and W R (positive)	Kahn and W R (doubtful)	Kahn and W P (negative)	Total cases	Percentage of W R (positive)
L3	14	4	16	34	41.2
L2	36	20	109	165	21.8
L1	24	14	161	199	12.0
Neural of all degrees	3	1	20	24	4.2

It will be seen that the positive rate decreases markedly from 41 per cent. in the advanced leprosy to only 4.2 per cent. in the neural group. Further in an analysis of twenty-four lepromatous cases which were discharged from the Institution four showed a strongly positive Wassermann on admission and when discharged the Wassermann reaction was found to be negative although no anti-syphilitic treatment had been given.

In our opinion these results give strong support to the theory that a positive Wassermann or Kahn reaction has no significance in leprosy and that physicians are not justified in giving anti-syphilitic remedies in leprosy unless there are clinical signs of past or present syphilis or a history of past infection.

It should be remembered that both the Wassermann and Kahn reactions need experience in order rightly to be interpreted. While the Kahn test is comparatively simple it should be only undertaken under reasonably efficient laboratory conditions and by those who have had sufficient technical experience to enable them not only to carry out the test but to interpret it accurately.

Lepromin Test

As far back as 1916 Kensuke Mitsuda, a Japanese worker, devised a skin test in the hope that it would aid in the diagnosis of leprosy. This test was first described by him at the Strasbourg Conference (1924) and subsequently taken up by Borgehr (1926) and Rogers (1930) brought the importance of the test to the knowledge of workers. Since this date Hayaishi (1933), Murr (1933) and others have reported on the test and it is becoming of increasing importance. Lepromin or more correctly leprumin is a saline emulsion of leprosy material with much of the extraneous tissue material separated and consists chiefly of a suspension of *M. leprae* with a certain proportion of tissue substance. It cannot be accurately standardised and therefore the dosage must be an arbitrary one—the usual dosage is 0.2 c.c. of the saline bacillary emulsion intradermally.

The following is the technique of preparing lepromin adopted at the Lady Willingdon Leprosy Sanatorium and based on the method originally described by Murr (1933).

¹ Only 5 per cent. of the serum reactions were with Kahn. 1 sera 1 of serum in per cent
 1) Wassermann test 1 was 1 tested

Material is obtained by trimming the ears of advanced lepromatous cases with pendulous or nodular ear lobes or by removing large soft nodules elsewhere on the body. Several suitable patients should be chosen so that a large amount of material may be collected at one time. The cuttings are boiled in water for twenty minutes and divided into fine fragments with a knife or scissors. The material is then dried for a few hours under a fan and thereafter in a vacuum desiccator over pure sulphuric acid. The dried material is ground up to a fine powder in a glass mortar and stored in a desiccator. This forms the stock powder. It is important in preparing the powder to make a large amount at one time so as to ensure as far as possible uniform results.

In preparing the suspension 0.4 grammes of the dry powder is ground up with 10 c.c. of saline. The fluid suspension is pipetted off. The solid residue in the mortar is again ground up with saline and the fluid suspension pipetted off and added to the rest of the suspension. This process being repeated three or four times. The whole suspension is then shaken up in a large test tube and allowed to sediment for ten minutes after which the fluid is again pipetted off, the sediment being discarded. Normal saline with 0.5 per cent. carbolic acid is added to make 100 c.c. The suspension is then made up in 1 c.c. ampoules which are sealed and heated at 120° C. for half an hour.

It is difficult to standardise lepromin accurately. This is shown by making a series of intradermal tests with full strength standard lepromin and dilutions of 1 in 2, 1 in 4, 1 in 8. While the stronger suspensions give a somewhat stronger reaction the difference in reaction is not in proportion to the difference in dilution. 1 in 8 giving a reaction not very much less than full strength. For the sake of uniformity however we keep a standard smear of each suspension which has been prepared by spreading out a standard loopful over a given area of slide and the concentration of bacilli compared. No attempt at counting the bacilli is made but it is possible to tell whether the numbers are approximately equal. By making several such examinations before diluting the suspension it is possible to adjust the strength by adding more or less saline.

The thumb nail on the anterior aspect of the forearm below the elbow is chosen for inoculation, the lepromin being injected. The usual dose is 0.1 c.c. Care should be taken that injection is made into and not under the skin. A wheal should rise round the point of inoculation. A slight serous effusion appears after the injection and disappears after a few days. In positive cases nodules appear at the sites of inoculation between the first and third week and increase gradually in size up to a maximum.

The degree of reaction is read by measuring the size of the nodule in millimeters between the blades of a sliding caliper. Readings are made after fourteen days and then once a week up to the eighth or tenth week. The maximum reading of each nodule is taken as the index. The following is a guide to the strength of the reaction.

Up to 3 mm in diameter	1+
3 to 5 mm in diameter	2+
Above 5 mm in diameter	3+
Nodule suppurating	4+

It is known that lepromin as prepared by the above method contains not only bacilli and the constituents of a nodule but tissue detritus as well. Hernandez (1940) has pointed out that in the lepromin reaction there are two phases, one an early reaction which takes place within twenty-four to forty-eight hours and a delayed reaction which

reaches its maximum in about four weeks. As a result of research work particularly by Lowe and Dharmendra (1943) the fractions which produced the early reaction were isolated and a refined lepromin made. This refined lepromin resulted in an early reaction which could be read within twenty four to forty eight hours and brought the test on a similar basis as the Mantoux test. Lowe and Dharmendra (1943) have disproved the contention that the two reactions are due to different substances or toxins and have shown clearly that the late reaction is due to the slow liberation of the antigen from the breaking down of the bacilli contained in the lepromin. From this the authors went on to isolate the specific fraction and have shown that as in tuberculosis so in leprosy the nucleo protein fraction in the bacillary emulsion is probably responsible for the specific reaction. For those who are interested in this work and the preparation of the newer refined lepromin they are referred to the original articles by Lowe and Dharmendra.

The question arises whether lepromin is a specific test for leprosy. In this connection it may be pointed out that Cochrane and Fielding (1941) undertook a study of three hundred medical students in Australia and found that all but three cases showed a negative lepromin. One of the three showed a moderately positive reaction on biopsy of the lesion typical giant and epithelioid cells were seen. In the other two the reaction was slightly positive. Dharmendra and Jaisaria (1941) however showed that with the old lepromin or as I term under terms it lepromin integra the percentage of positives in a population free from leprosy was as high as 60 per cent and that with the refined lepromin the percentage was reduced to 3 per cent and expresses the opinion that the work done so far indicates the existence of a specific antigen in the leprosy bacillus. The discrepancy between the results of Cochrane and Fielding and Jaisaria and Dharmendra can probably be explained by the fact that they were dealing with a different racial group.

It is however generally agreed that while the refined lepromin is more accurate because its preparation needs a fully equipped laboratory for all practical purposes lepromin integra or whole lepromin can be used.

The Significance of the Lepromin Test

It must be very clearly understood that the lepromin test is neither a diagnostic nor a therapeutic test. As was pointed out in the previous chapter all reactions against the M. leprae are defensive in nature and that successful defence is shown by the ability of the tissues to anchor the organism and prevent its dissemination from the skin to the reticulo endothelial system. The lepromin test is merely an indication of the actual or potential capacity of the body to put up a successful defence. Therefore as can be expected in all tuberculoid cases the test is positive and its degree of positivity is in direct proportion to the intensity of the tissue reaction. In all lepromatous cases the reaction is negative. As already indicated there are a group of cases not altogether understood in which the defences of the body are thrown into complete confusion. These cases represent partially successful defence. The lepromin test in these cases shows considerable variation. Most frequently it is negative but during phases of acute exacerbation the test may be slightly to moderately positive which may either become negative in the quiescent phase or less positive. While in all probability this test indicates allergy or potential allergy we prefer to state that a positive lepromin shows successful or potentially successful tissue defence and we

I believe that a lepromin reaction (excluding the reaction produced by extraneous tissue material) can only be positive in the presence of a primary focus and therefore is analogous to the Mantoux test. This opinion receives support from the observation that we have never succeeded in producing a positive lepromin in a monkey without previously inoculating the animal with a nodule from a patient with leprosy.

If then the lepromin reaction represents a positive tissue defence what causes a reaction to be negative? Cochrane Rajagopalan *et al* (1941) showed that (a) 86 per cent of the healthy adults examined showed a positive lepromin and 79.6 per cent of healthy children and (b) that the degree of positiveness varied according to the type of the cases. They further showed that 60 per cent of the so called simple macular and 82.2 per cent of the tuberculoid macular lesions of neural leprosy were positive.¹ On analysis of the lepromin reaction in relationship to known history of contact it was shown that the percentage of positive reactions was in direct proportion to the closeness of contact with an open case and the following conclusions were arrived at:

(a) In healthy persons the older age group tends to show a higher percentage of positives. How far this is due to small subminimal infection with *M. leprae* it is impossible to say owing to the paucity of the numbers and to the absence of information from non endemic areas. Is this one of the reasons why adults are less susceptible to leprosy than children?

(b) In children the positive reactors appear to decrease as the closeness of contact increases.

(c) It is our opinion that the more intimate the contact in early life the more likely is cellular resistance to be broken down. If it could be established that lepromin negative children more frequently become lepromituous in adult life this would be of help in the campaign against leprosy.

In conclusion therefore it may be said that the lepromin test is of value in the prognosis of leprosy but is of no help in its diagnosis. A positive lepromin indicates the actual or potential ability of the tissues of the body to put up an effective defence against the *M. leprae*. It is of farther help in the classification of the disease for it is of some assistance in aiding the clinician in separating the true major tuberculoid lesions from the large group of atypical or border line cases which will be described in a subsequent chapter.

¹ For explanation of these terms see Chapter VI





CHAPTER VI

SIGNS AND SYMPTOMS

From what has been written in the previous chapters it will be clear to the reader that leprosy naturally falls into two distinct types (i) neural leprosy (ii) lepromatous leprosy. In this chapter it is proposed to describe in detail the signs and symptoms of leprosy. It should be said at the outset that every practitioner who is faced with the possibility of making a diagnosis of leprosy should only do so after very careful and detailed consideration. A useful maxim which should not be forgotten especially when dealing with children is: When in doubt never diagnose leprosy. It may be thought that if a diagnosis is not made one is condemning the patient to a greater possibility of developing more serious lesions and decreasing the patient's chances of effective recovery. In this connection it may be said that there is little evidence to indicate that delayed treatment except in the lepromatous case which should not be missed has any effect on the subsequent progress or retrogression of the disease. Further it will be contended in the chapter on the development of the lesions of leprosy that apart from a very limited group the various types of leprosy remain true to type and do not change. If in addition one remembers the great tendency for early neural lesions to disappear the statement that one should think many times before diagnosing leprosy appears justified. In fact to make statements such as 'You have suspicious signs of leprosy' is not only unjustifiable but definitely harmful for until the outlook towards leprosy is radically changed one must remember the psychological and spiritual distress which is caused when such a diagnosis is made. On the other hand when the signs are unequivocal then the diagnosis need not be hidden under pseudonyms such as Hansen's disease. Let us treat the patient as an intelligent being not alarm him when there is no need for alarm but when a diagnosis is certain encourage him to adopt a sane and reasonable attitude towards the disease and thus create in him that determination and unity of outlook which will result in the greatest amount of co-operation between the patient and his physician. These remarks with regard to diagnosis apply with still more force when a physician is faced with the possibility of leprosy in children.

In the light of the statements it is emphasised that a diagnosis of leprosy except in one connection which will be mentioned later is never justified unless one of the two cardinal signs are present:

- (i) Clinical signs of nerve involvement
- (ii) The demonstration of M. leprae in the skin and frequently also in the nasal mucous membrane

I Neural Leprosy (symbol N)

We now pass on to the detailed description of neural leprosy. Generally speaking, neural leprosy is benign and resistant showing a marked tendency to spontaneous healing and is divided into the following subtypes:

- (i) Simple macular leprosy (symbol N₁)

(ii) Neural tuberculoid leprosy

(a) Minor tuberculoid (symbol Lt)

(b) Major tuberculoid (symbol NT)

(iii) Neural anaesthetic leprosy (symbol Na)

(i) *Simple Macular Leprosy (Neural type) (Na)*

This type of leprosy manifests itself as single or more usually multiple hypopigmented lesions which are commonly found

(a) on the face

(b) extremities (lateral aspects)

(c) buttocks

(d) scapulae



FIG. 28—Simple neural lesions showing typical macules on back of arm and shoulder



FIG. 29—Simple neural lesion showing macule on thigh. Note well defined edge

The lesions are hypopigmented (not depigmented) with a well defined edge the edges are definite and show a pronounced demarcation between the affected and healthy skin but are not raised. In other words the macules in simple neural leprosy are macules in the dermatological sense. In addition the macules especially on the extremities and buttocks usually feel dry to the touch and frequently show in a greater or lesser degree anhydrosis or lack of sweating. Associated with the dryness of the lesions there may be particularly on the skin over the tibia a patterned appearance of the skin with slight scaliness indicating irregular cornification or parakeratosis. Another characteristic of the hypopigmented lesions of simple macular leprosy is loss of sensation. This

may not be seen in all the macules but it is constantly found in some. It should be noted that

(a) Patches on the face usually show no loss of thermal, tactile or pain sensibility.

(b) Patches on the trunk not infrequently show no anaesthesia to touch but loss of thermal sensibility.

(c) Patches on the extremities generally show anaesthesia to touch as well as to heat.

In this type of leprosy it should be borne in mind that unless lesions on the face are absolutely typical no diagnosis should be made. Further the lesions are nearly



FIG. 30.—Typical hypopigmented lesion on the buttock.



FIG. 31.—Slightly erythematous lesion on the right knee shows the fine granular appearance of very early macular leprosy.

always multiple and great hesitance should be shown in making a diagnosis unless anaesthesia is definitely present remembering that loss of thermal sensibility is the earliest sign to appear. If in doubt a biopsy of the lesions should be made. In this connection two points should be borne in mind.

(a) In taking a biopsy care must be exercised to include in the section a piece of subcutaneous fat. It is changes in the corium and in the small subcutaneous nerve which must be seen and unless subcutaneous fat is included it is frequently impossible to make a diagnosis.

(b) Fix the tissue in Zenker's solution rather than formalin—the latter tends so to distort the cells that it is almost impossible to make an accurate diagnosis.

While in many instances the histology of the simple macule is not characteristic if the round-celled infiltration is concentrated around hair follicles and vessels in the corium

and the small nerves in the skin are seen to be invaded by the same process then the lesion is most probably one of leprosy. If in addition there are foci of epithelioid cells with round celled infiltration then the diagnosis is almost certainly confirmed.

(ii) *Neural Tuberculoid Leprosy*

Wade and Lowe have pointed out that the simple macule of neural leprosy is essentially tuberculoid in character. That is histologically the same changes are seen the difference being rather a matter of degree than of character.

All lesions of tuberculoid leprosy show themselves as raised infiltrated lesions



FIG 3



FIG 33

Especially minor tuberculoid lesions. Note the pebbled described first by Wade

and therefore are not macules in the dermatological sense. They are divided for convenience into two subtypes

- (a) *Minor tuberculoid*
- (b) *Major tuberculoid*

(a) *Minor Tuberculoid (Nt)* With regard to the minor tuberculoid division of neural type leprosy it may be stated that it is generally accepted that all active lesions of neural leprosy will show histologically tuberculoid change.

The main difference clinically between the simple macule of neural type leprosy and the minor tuberculoid macule is that the former is flat with a definite but not a raised edge and the latter may be raised or at least the periphery is infiltrated. The main characteristics of the lesions are as follows

- (1) The lesions are usually hypopigmented, the edge slightly erythematous, always shows a varying degree of infiltration which may be uniform or present as characteristic pebbled appearance.
- (2) The distribution of the lesions is similar to those of simple macular leprosy.

- (3) Not infrequently in addition to the lesion being infiltrated if carefully examined the superficial cutaneous nerve supplying the area in which the lesion occurs is found to be enlarged and sometimes tender
- (4) Loss of superficial tactile sensation is usually constant accompanied also by loss of thermal sense and frequently lack of appreciation of pin prick sensation
- (5) As a rule lesions of simple macular leprosy as well as those of minor tuberculoid leprosy are negative to standard methods of examination. Occasionally and this it is said applies particularly to the African races a few bacilli may be possible of demonstration. This emphasises the fact that it is not the presence of the bacilli that determines the classification of leprosy but the clinical signs



FIG 11 — (Tubercle) lesion of the right cheek



FIG 12 — (Tubercle) lesion of the right cheek

(b) *Major Tuberculoid (NT)* In essential characteristics there is no difference between major and minor lesions. This classification is adopted for convenience but the signs and nature of the lesions are essentially the same the difference is seen in the degree and not in the nature of the changes. Major tuberculoid leprosy is seen in individuals in which there is an active and sometimes violent tissue response on the part of skin and nerves to the presence of *M. leprae*. The following are the chief clinical signs

- (1) Raised erythematous and grossly infiltrated lesions. Infiltration is not usually confined to the edge but the whole patch is raised forming a plaque
- (2) Edges are well marked and grossly infiltrated
- (3) Loss of tactile thermal and pin prick sensibility. This is frequently seen at the centre which often shows an area of normal skin
- (4) There may be enlargement of any of the superficial nerves. Frequently the small branches of the cutaneous nerves e.g. the superficial branch of the radial nerve crossing the lower end of the radius may become enormously

enlarged The enlarged nerve can usually be traced to an erythematous infiltrated lesion although the enlargement may persist long after the patch has disappeared

- (5) The distribution is similar to the lesions of neural type leprosy in general. It must be remembered that major tuberculoid leprosy invades the so called immune areas more often than the other neural lesions. The areas are: axilla, eyelids, vertebral column, palms of hand.
- (6) In major tuberculoid leprosy, especially in the more acute forms where there is marked erythema, the lesions are usually positive to standard methods of examination, but the finding of bacilli is not a reason for any alteration in classification.



FIG 36—Spreading major tuberculoid lesion on back. Several enlarged nerves could be palpated.



FIG 37—Major tuberculoid lesion on dorsum of hand. Note wrinkled appearance—indication of resolution.

- (7) It should be borne in mind that major tuberculoid lesions are sometimes unilateral. This is frequently so on the face when one side of the face or ear and not the other is affected. This is an important point in differentiation between these lesions and the more serious lepromatous lesions which will be shortly described. In all major tuberculoid lesions there is a tendency for them to show signs of acute exacerbation. These will be described in the chapter on reaction in leprosy.

(iii) Neural Anaesthetic Leprosy (N₂)

This subtype of neural leprosy is not so commonly seen in South India as in North India and is that type of leprosy which manifests itself in polyneuritic changes without macular lesions in the skin. The lesions appear to be purely neural and as mentioned in the previous chapter are visualised as caused by bacilli passing from the primary focus to the nerves rather than to the skin and setting up a fibrous tissue reaction which results in the destruction of the nerve. This fibrous tissue reaction is evidently not only confined to the larger nerves but there appears to be a fibrosis of

the nerve-endings in the skin resulting in a true peripheral neuritis. The latter change has never been demonstrated but it is difficult to explain the extensive glove stocking anaesthesia in any other way especially as it is a dissociated anaesthesia and not a complete loss of sensation. Muir and others have suggested that it is a true ascending neuritis but such a theory is a difficult one to prove. Gross changes have not been described in the cord although bacilli have been demonstrated in the anterior horn but these we believe are of no significance. In the field of neurology there is yet much research work to be done into the actual cause of lesions of neural anaesthetic leprosy but this could only be taken up by one well versed in the technique of demon-



FIG 34—Major tubercles on the hand showing the typical nodular character



FIG 35—Major tubercles on the buttock showing the typical nodular character

strating changes in the peripheral nerves and in the nerve-endings of the skin. With these introductory remarks we now pass on to the signs of neural anaesthetic leprosy. These are as follows:

- (1) Anaesthesia
- (2) Nerve enlargement
- (3) Muscular paralysis

(1) *Anaesthesia* This as has already been indicated is one of the cardinal signs of all neural type leprosy and should be looked for in every case. In neural anaesthetic leprosy loss of sensation is confined to the extremities especially to the areas of distribution of the common peroneal and ulnar nerves although the whole of the foot and leg or hand and arm may be affected giving rise to the well known glove stocking anaesthesia described by Muir. It should be remembered that loss of sensation is usually seen in the following order: thermal, tactile, pain, pressure.

(2) *Nerve Enlargement* This is most marked in tubercular muscular leprosy where in addition to gross enlargement of the subcutaneous nerves to the cutaneous

lesions there is seen marked enlargement and not infrequently abscess formation in the ulnar common peroneal and great auricular nerves. In neural anaesthetic leprosy however as a result of a fibrous tissue reaction enlargement of the ulnar and peroneal nerves may be seen. In the early stages pain and tenderness are prominent symptoms but later the nerves are reduced to fibrous strands this is due to contraction of the fibrous tissue then the nerve may be actually less thick than normal. The amount of sensation lost is dependent on the degree of nerve destruction. In the early stages of enlargement and tenderness only anaesthesia to temperature and touch may be present but as fibrosis sets in pressure sensibility—both superficial and deep—is affected. It is interesting to note that the deep reflexes such as the knee jerks are never lost and that deep pain is seldom affected neither is joint sense.



FIG. 40.—Major fibrotic leprosy. (Note hand deformity due to a cicatrix.) Photo taken by the Photography Department General Hospital Madras.

(3) *Muscular Paralysis Paresis and Wasting* Accompanying the enlargement of the ulnar and common peroneal or later due to secondary fibrosis the characteristic wasting of the hand as seen in claw hand is a common sign. A similar wasting of the muscles supplied by the common peroneal is noted with accompanying drop foot. In addition the VIIth nerve is not infrequently involved resulting in facial paralysis and lagophthalmos. Accompanying VIIIth nerve involvement Vth nerve lesions may be seen in anaesthesia of the cornea. In passing it may be said that both drop foot and facial paralysis seldom recover but by judicious exercise and massage and by preventing disuse atrophy a considerable amelioration of symptoms due to claw hand can be effected.

Trophic Conditions Neural anaesthetic leprosy is one of the diseases which show marked trophic changes. These are

all due to nerve involvement leading to the alteration of the vascular supply to the limbs and also partly the result of pathological changes in the arteries and veins especially the veins resulting in disturbances of nutrition producing a local loss of calcium. The earliest lesions seen are frequently in the form of idiopathic blisters or injuries due to the fact that patients do not appreciate pain from burns or slight injuries. It is interesting to note that osteoporosis is noted without trophic ulceration and in cases showing claw hand rarefaction of the phalanges and metacarpals may be seen. This may go on to actual bony absorption as is witnessed by the fact that in such cases traces of the finger nails can be demonstrated on the shortened stumps. It is our opinion that actual necrosis of bones and signs of periostitis and irregular ossification are never seen in leprosy apart from sepsis resulting from trophic ulceration.

The trophic ulcer is a well known condition and is seen in leprosy diabetes and syphilis but in the latter two conditions bone is seldom so extensively involved. We

believe that all trophic ulcers commence from injury. This may be so slight as hardly to be noticed but in an anæsthetic foot it is sufficient to cause pathological changes. The commonest site for trophic ulceration is the heads of the metatarsal bones especially the first and fifth. As a result of pressure areas of cornification are seen and later a definite ulcer forms which gradually extends down to the bone. Sepsis results and bony necrosis sets in. Trophic ulceration of the toes is also frequently seen as a result of injuries—e.g. from closely fitting shoes or injuries from stones and again these become septic and result in necrosis of phalanges subsequent sequestra formation and extrusion of the dead bone through the ulcer. The larger bones such as the metatarsal may be affected causing extensive trophic ulcerations which do not heal until the whole bone is removed. It is interesting to note that while the phalanges of the hand are frequently



FIG. 41—Major trophic ulceration back of right femur. Necrosis of heel after edge.



FIG. 42—Tube insertion on left cheek. Fungus element of growth.

found to be necrotic necrosis is seldom seen in the metacarpal bones. This is because the carpal bones are far less liable to injury than the metatarsal bones. The latter along with the tarsal bones are exposed to injury because they have to bear the whole weight of the body as in walking and standing.

Occasionally both the phalanges and metatarsal bones escape involvement and the tarsal bones are affected. This usually results from trophic ulceration of the heel leading to necrosis of the os calcis. The trophic condition may extend and a condition similar to a Charcot joint is seen.

Neural anæsthetic leprosy is one of the most mutilating forms of leprosy and while the bacilli cannot be demonstrated by standard methods of examination yet the resultant fibrosis of the nerves may give rise to extensive changes and may cause not only advanced trophic changes but double facial palsy, corneal ulceration, drop foot and marked muscular wasting giving rise to one of the most pathetic sights in the whole realm of chronic diseases.

The reader in studying this chapter on signs of neural leprosy will realise that simple macular leprosy as well as tuberculoid macular leprosy may be accompanied by *inaesthesia of the extremities with muscular paralysis*—e.g. claw hand and drop foot and trophic ulceration. While these last signs are particularly noted in neural anaesthetic leprosy in classifying neural leprosy we reserve the designation neural anaesthetic leprosy only to those cases which show polyneuritic changes without accompanying skin lesions. Where such lesions are seen associated with macules then the classification is determined according to the nature of the macules whether they are of a simple neural or tuberculoid type.

In passing it might be noted that the word *macule* is used in the broad sense by leprologists and not confined only to lesions not raised above the surface. If the term *macule* were used according to the strict dermatological usage the classification of leprosy would become altogether too complicated.

It probably would be more in keeping with modern dermatological opinion if the term *macule* were reserved for the simple neural lesion and the term *lepride* were used for all tuberculoid lesions for the latter lesions histologically are in the nature of leprides and somewhat comparable as Wade has pointed out to the tuberculide and syphilide. However it has been felt that the standard classification laid down at the Cairo Conference (1938) still represents to date the most satisfactory description of neural leprosy and therefore this has been closely followed.

The School of Tropical Medicine Calcutta adopt a modification of the Cairo Conference classification. All neural lesions which show macules are classified as *neuro macular lesions* and are designated by the symbol *Nm*. If there are polyneuritic signs in addition the suffix *a* is attached and so neural lesions with macules



FIG. 43 — Major tuberculoid leprosy. Plaque like lesion on the left back. One or two of the smaller lesions are nodular but retain the flat topped characteristics of major tuberculoid leprosy.

and polyneuritic signs would be designated *Nma* and pure neural anaesthetic cases would remain with the designation *Na*. The term *neuro macular* as a separate classification has not been recommended by the Cairo Conference therefore we have not followed this classification. Further we feel it is important to differentiate between simple neural lesions and neural tuberculoid lesions and therefore are of opinion that the description of the neural lesions as above described = the simplest and the most practical.

II Lepromatous Leprosy (non-resistant or malignant) (symbol L)

Previous to the Cairo Conference (1938) the nomenclature of this type of leprosy was not satisfactory. Prior to the rediscovery of the Chaulmoogra derivatives this type went under the general name of nodular leprosy. Muir and Rogers called it

cutaneous leprosy. Neither of these terms however were satisfactory, the former because nodules are only a late manifestation of this type of leprosy, the latter because cutaneous signs are not solely confined to cutaneous leprosy but also occur in neural leprosy. For the reasons the Cairo Conference (1935) classified this type of leprosy under the general name 'lepromatous'. This designation is more satisfactory for it recognises the basic histology of the lesions, the presence of lepra cells and indicates that this type is serious and non resistant. Further characteristics of lepromatous leprosy are that it is always positive to standard methods of bacteriological examination and all cases show a lepromin negative reaction. The Cairo Conference did not further subdivide lepromatous leprosy because it was felt that in contrast to neural leprosy



Fig. 44. A clinical picture of lepromatous leprosy showing the characteristic nodules and ulcers on the hands.

there were no true or distinct subtypes. While we believe this to be the case, yet there is still a great deal of confusion as to the forms and varieties of lepromatous leprosy and we believe that while all lepromatous leprosy is essentially the same that is clinically plus if this type tend to pass into each other yet certain varieties seem to have a better prognostic significance than others but not until a much more detailed study of lepromatous leprosy is made will it be possible further to unravel the tangle which we feel still surrounds the various manifestations of lepromatous leprosy. It may however be said that leprosy clinically falls into the following subdivisions:

- (a) Macular lepromatous leprosy
- (b) Diffuse lepromatous leprosy
- (c) Infiltrative lepromatous leprosy
- (d) Nodular lepromatous leprosy

(a) *Macular Lepromatous Leprosy*

When the question of the development of lesions of leprosy is under discussion it will be pointed out that it is our belief that lepromatous leprosy does not commence as lepromatous but generally develops either from certain simple macules of neural type leprosy or from for want of a better term what might be called the prelepromatous macules which we believe in especially children are a definite clinical entity and have been styled incipient lesions of childhood. Macular lepromatous leprosy then shows itself in the form of multiple hypopigmented erythematous and usually slightly infiltrated macules. These macules vary from those described under neural leprosy in three important aspects

- (i) distribution
- (ii) size
- (iii) appearance

(i) *Distribution* The simple macular lesions of lepromatous leprosy show no tendency to be largely confined to the face, buttocks, outer aspect of extremities, scapulae but are usually diffusely distributed over the body.

(ii) *Size* Generally speaking the lesions of simple macular leprosy are smaller and very much more numerous.

(iii) *Appearance* In the macules of lepromatous leprosy the periphery is not well marked but fades imperceptibly into the surrounding skin. In other words unlike the simple neural macule there is no marked demarcation between the normal skin and the affected skin of the macule. Further while the simple macule of neural leprosy tends to be dry and of a different texture and may show anhydrosis the lepromatous macules on the other hand show no difference in texture from the normal skin exhibit no lack of sweating and have in addition a slightly shiny (erythematous) appearance. Although the infiltration is not obvious on careful examination these macules usually show some degree of infiltration although it may be very slight. The indefiniteness of the edge and the lack of tissue reaction results in no marked colour contrast between the affected and normal skin. Frequently owing to slight erythema these lesions are better seen in an oblique but bright light when occasionally the macules appear grey looking. The simple macule of lepromatous leprosy is always positive to standard methods of examination. Owing to the vagueness of the lesions and the indistinctness of the periphery these lesions are much more difficult to recognise than the macules of simple neural leprosy.

(b) *Diffuse Lepromatous Leprosy*

While it has been held that this is a true type of lepromatous leprosy we are of opinion that diffuse lepromatous leprosy is usually the result of the gradual coalescence of the numerous vague macules of macular lepromatous leprosy until the whole skin is involved in a generalised lepromatous infiltration. These cases in the earlier stages are very difficult to detect because no actual lesions are demonstrable. The skin generally has a shiny appearance and there is slight infiltration but this is more easily appreciated by touch rather than by sight. The infiltration is better detected by picking up the skin of the back between the fingers. In such cases when examined carefully signs slightly more definite are usually found on the face. The ear lobes are usually



FIG 45



FIG 46



FIG 47



FIG 48

Less in of u aculi p rono to leg rony (Not the n h pl ts of the l s ns, the n lot n throa
of th periphery n th r l tr l t A B placed in th centre of th micules on foot
a l l l n n

slightly thickened and shiny and the eyebrows may show some thinning or loss of hair. It is however to be remembered that loss of the eyebrows is a late sign in lepromatous leprosy. The ear lobes are not infrequently quite markedly thickened when the lesions are much less conspicuous elsewhere and it is wise to ask the patient to turn round and stand straight for thickening and slight nodulations of the ear lobes can often best be appreciated by standing facing the patient's back.

(c) *Infiltrative Lepromatous Leprosy*

Lepromatous leprosy as will now be realised may show itself in macules diffuse infiltration areas of marked infiltration or nodules and infiltrative lepromatous leprosy is merely a more advanced stage of macular lepromatous leprosy. The infiltra-



FIG. 40.—Macular lepromatous leprosy showing activity of lesions—raised and erythematous—but the edge of the lesion tends to be less marked than the centre.

tion may appear in the hypopigmented patches or develop in the skin which shows diffuse lepromatous leprosy. The characteristics of lepromatous leprosy remain that is the edges while raised do not stand out as in tubercloid leprosy but gradually shade off into the surrounding skin and do not show the sharp line of demarcation of neural leprosy. Further the infiltration is not of a whip cord like feel but is much softer or as Wade describes it more succulent. These points are worthy of mention because for those with little experience such differences need to be described for confusion in classification can arise if one does not carefully observe the type and nature of the infiltration. It must be remembered that the infiltration of lepromatous leprosy may show great variation of consistence texture and in the lighter rice colour and therefore the clinician is advised carefully to study the varying clinical manifestations.

(d) *Nodular Lepromatous Leprosy*

Nodular leprosy we believe does not arise *per se* but always is the result of progressive deterioration of the macular diffuse or infiltrative forms of lepromatous leprosy. In the earlier stages nodules appear first on the ears later as the disease advances they may appear anywhere on the body and are commonly seen on the extremities especially the elbows fingers over the joints and sometimes on the genitals in the male and on the vulva in the female. By the time the lepromatous leprosy has become so advanced as to reach the nodular stage diagnosis should no longer be in doubt. At first many of the nodules tend not to be fixed to the skin and are movable in the subcutaneous tissues but later they become fixed and are liable to ulcerate. It is the ulcerating nodular case which presents a very great problem in nursing.

In lepromatous leprosy the nasal mucosa is frequently positive to standard methods of examination and nasal as well as eye lesions are a common complication. A detailed description of these lesions will be given in a later chapter. While some



Fig 50



Fig 51

Difference from to 11 proxy Note lipoma on the back and the loss of eyebrow in Fig. 51



Fig 52



Fig 53

Infiltrative leprosy not well proxy Note the erythema of skin into the area in ling skin in contrast with infiltration in the back to 11 proxy (see Figs 30-43)

authorities state that the scalp is not affected in leprosy in certain races particularly the Mongolian and European a leprous alopecia is sometimes seen The whole of the hair of the head falls out except a thin line of hair following the great vessels of the scalp—temporal and occipital In addition actual nodules may form on the scalp itself

In the above description of lepromatous leprosy no mention has been made of neural signs Just as neural leprosy is not only confined to the nerves but shows cutaneous lesions so lepromatous leprosy is not solely confined to the skin The nerves are invariably involved in lepromatous leprosy that is if carefully examined e.g. scrapings actually taken from nerve sheath—the bacilli can be found inside the nerve bundles As explained however in the chapter on Pathology these bacilli lie within the nerve sheath without at first causing much damage and frequently no loss of



Fig 54

Nodular lepromatous leprosy



Fig 55

sensation can be detected The bacilli however ultimately act as foreign bodies first causing some swelling and oedema of the nerve and this later results in an interstitial fibrosis therefore frequently in lepromatous leprosy depending on the amount of pressure as a result of oedema or fibrosis nerve fibres are affected and are liable to be destroyed anaesthesia is then elicited and this as in neural leprosy usually involves the distribution of the common peroneal or ulnar nerves As the disease progresses lepromatous leprosy shows both neural and cutaneous signs the word mixed however is not used first because all leprosy is essentially mixed that is there are both nerve and skin manifestations in neural as well as lepromatous leprosy the neural anaesthetic case being an exception Again mixed leprosy is not a separate clinical entity or a different type and therefore if a case is lepromatous it is classified as such without reference to neural involvement but in symbolising the classification the letter N is added to indicate the neural as well as the lepromatous elements

Secondary Neural Leprosy

As far back as the latter part of the nineteenth century Hansen, Impey and others recognised the self healing nature of leprosy and Muir likened the course of leprosy to a parabolic curve commencing at the base line with neural leprosy passing through to the lepromatous stage reaching a peak and then gradually the bacilli disappear from the skin and finally reaching the base line again and as Muir termed it burning out leaving residual gross nerve lesions. While it is perfectly true to state that leprosy is a self healing disease it is not correct we believe to visualise the disease normally passing from the neural stage to the lepromatous and finally burning out as an advanced neural case. It is our conviction that the various types of leprosy as a rule remain true to type that is neural remains neural except for a small group of cases which will be referred to in a later chapter and



FIG. 6.—Diffuse leprosy gradually passing into the infiltrative and nodular type. Note the not uncommon freedom of the nose on this type compared with the face.



FIG. 57.—A bundle of bacilli, some dead but how the bacilli act in between the bundle which has become organized into fibrillar tissue (interstitial fibrosis). This type of bacillar action occurs early in neural leprosy. It is a late action in leprosy. (x 100)

leprosy starts with what may be termed

the prelepromatous macule which passes gradually into fully developed lepromatous leprosy. In the great majority of instances however lepromatous leprosy except when it is arrested either as a result of treatment or much more rarely spontaneously remains lepromatous throughout life. While one states this it must be remembered that as lepromatous leprosy advances so the resultant interstitial fibrosis in the nerves increases and thus as the disease progresses more and more advanced signs are seen and deformities absorption of bones and more rarely paralysis may all be an accompaniment of lepromatous leprosy. In a few instances however as the secondary nerve signs develop the bacilli begin to disappear from the skin and the skin lesions become flattened leaving a wrinkled or as Muir termed it tissue paper appearance of the skin and gross nerve deformity. It is these cases which are termed secondary neural leprosy. There has never been any adequate explanation as to why the bacilli disappear from the skin

It is our belief that there must be some alteration in the biochemical or biophysical state of the skin which effects its nutrition and as a result the bacilli can no longer multiply in the skin therefore the disease as we have said ceases to progress because we believe that leprosy can only develop as a result of the bacilli passing from the primary focus to the skin and then throughout the reticulo endothelial system Some support is given to this statement (1) because bacilli have been described in internal organs after they have disappeared from the skin (2) because if a patient is starved almost to the point of death or becomes emaciated and cachectic bacilli tend to disappear from the skin only to reappear if the nutrition of the skin returns more nearly to normal





CHAPTER VII

CLASSIFICATION AND TECHNIQUE OF EXAMINATION

It is unnecessary to review the history of the various attempts at classification. It is sufficient to mention the fact that the present classification is primarily a clinical one. In classifying a case the more important conditions are noted for instance a simple macular case may show polyneuritic signs but in so far as the most obvious and prominent lesions are the hypopigmented patches it would be placed in the simple neural category. Again it must be borne in mind that the present classification is still an interim one and therefore it is not always easy definitely to label every case. Hence the histological examination and the lepromin test are useful adjuncts to classification but must only be used as aids and not as a basis for classification.

THE CLASSIFICATION OF LEPROSY ABSTRACTED FROM THE REPORT OF THE SUB COMMITTEE ON CLASSIFICATION (CAIRO CONFERENCE 1938)

The problems of classification of cases of leprosy should be viewed broadly bearing in mind both (a) the requirements and circumstances of work of the practical field worker to whom classification is necessary for purposes of prognosis treatment or control but who cannot apply elaborate or time consuming methods of differentiating type of the disease and (b) the refinements of such differentiation that are possible to the specialist who employs special methods of investigation. The great majority of persons who deal with leprosy work under circumstances that require that the basic or primary classification be as simple as possible.

Progress in knowledge of the forms of leprosy and of the nature of the leprosy processes has been made since the classification that is now most generally used was adopted by the Leonard Wood Memorial Conference on Leprosy in 1931 and it is now possible to modify some of the terms of that classification to eliminate certain causes of misunderstanding. However our knowledge of the matter has not yet progressed to a point where it is possible to attain unanimity of opinion on certain essential features.

It is recommended that for the present the basic division of leprosy into two types along the lines laid down in the Memorial Conference classification be continued until such time as further study of the matter permits the attainment of unanimity. It is further recommended that future research be in the direction indicated by the questions raised by the minority of this Committee the main questions being whether or not the neural type of the Memorial Conference classification should be divided into two distinct main types simple neural and tuberculoid. For the present it is the predominant opinion that such divisions should be considered as subtypes or varieties.

Objections have repeatedly been raised to both of the current names of the two types (i.e. neural and cutaneous) because of confusion arising from the special sense in which they are employed in leprosy classification on account of difficulties of translating them into other languages and for other reasons. However no other

words have been proposed which are free from similar objections. It is the opinion of the Committee (a) That for the time being at least the word neural should be retained for the type to which it is now applied (b) that because cutaneous has proved particularly confusing its use should be discontinued and replaced by the term lepromatous the symbol of which is L.

It is proposed that the definition of the two types of the Memorial Conference classification be amended as follows

PRIMARY CLASSIFICATION

Neural (N) Type

All cases of the benign form of leprosy with disturbances of polyneuritic nature (i.e. alterations of peripheral sensation trophic disturbances atrophies and paralyses and their sequelae) or macules of non lepromatous nature (i.e. leprides usually with localised sensory disturbances) or both. These cases give evidence of relative resistance to the infection are of relatively good prognosis as regards life although mutilation may take place and usually react positively to lepromin¹. Bacteriologically the skin lesions are typically but not invariably found negative by standard methods of examination though the nasal mucosa may be found positive. Many of these lesions are histologically of tuberculoid nature.

Lepromatous (L) Type

All cases of the malignant form of leprosy relatively non resistant and of poor prognosis usually negative to lepromin exhibiting lepromatous lesions of the skin and other organs especially the nerve trunks. Bacteriological examination usually reveals abundant bacilli. Disturbances of polyneuritic nature may or may not be present they are usually absent in the earlier stages and present in the later stages of primarily lepromatous cases and often present in cases arising secondarily from the neural form.

SUBCLASSIFICATION

Subdivision of the types of leprosy may be made from two points of view (a) with respect to the degrees of advancement of the disease and (b) with respect to the forms or varieties of cases within a type (i.e. subtypes) based on the nature of the lesions. The former method of subdivision is that of the Memorial Conference classification and it has proved useful in the hands of many workers especially in dealing broadly with large numbers of cases. The latter method of subdivision is generally employed in dealing more precisely with individual cases. Both methods have their uses and should be understood but no generally applicable practical formula for combining the two has not been arrived at. The two methods are dealt with independently.

I General Subclassification (by degrees of advancement)

The following specifications are unavoidably somewhat crude but they indicate in a general way the basis of the division into three degrees of advancement of each type

¹ Now termed lepromin

Neural 1 (N1)

Slight neural This includes (a) cases with from one to several small macules or a proportionally smaller number of larger macules whether flat or elevated without indications of polyneuritic changes or (b) cases presenting only polyneuritic changes of slight degree disturbances of peripheral sensation affecting one or two extremities not of marked extent with only minor trophic disturbances muscular atrophy or paresis if any or (c) cases showing combinations of macular and polyneuritic manifestations in equivalent degree of total affection

Neural 2 (N2)

Moderately advanced neural This includes (a) cases with fairly numerous or large macules of wide distribution without evidence of polyneuritic changes or with such manifestations of fairly slight degree or (b) cases presenting only polyneuritic changes of moderate degree peripheral anaesthesia of considerable extent if affecting only one extremity or less extent if affecting more than one and moderate trophic changes atrophies and paralyses including beginning contractures of limited extent or (c) cases showing combinations of equivalent total degree

Neural 3 (N3)

Advanced neural This includes (a) cases with very numerous or very extensive macular lesions of the more marked kind with polyneuritic changes or (b) cases presenting only advanced polyneuritic changes extensive peripheral anaesthesia and more or less marked motor and trophic disturbances paralyses atrophies contractures trophic ulcers and mutilations or (c) cases showing combinations of equivalent total degree

Lepromatous 1 (L1)

Slight lepromatous Cases with lepromatous skin lesions consisting of one or a few macular areas or a few small infiltrated patches or small nodules or diffuse lepromatous changes of slight degree lesions of the nasal mucous membrane are usually absent

Lepromatous 2 (L2)

Moderately advanced lepromatous Cases with numerous macular areas or fairly numerous small or fewer large infiltrations or nodules or diffuse lepromatous changes of moderate degree lesions of the nasal mucous membrane are frequently present

Lepromatous 3 (L3)

Advanced lepromatous Cases with numerous and extensive or very marked lepromatous lesions which may vary in their stage of development or retrogression lesions of the nasal mucous membrane are almost always present

Mixed Cases

Recognition should not be given to mixed leprosy as a type. However cases of the lepromatous type usually exhibit sooner or later varying degrees of polyneuritic involvement and for precision such mixed or complete cases may be designated

LN The symbol L should be given precedence, regardless of the original nature of the case or the relative severity of the two elements because of the predominant importance of the lepromatous element. In grading the degree of advancement of these cases the appropriate figure is placed after each symbol e.g. L2-N1 or L1-N3

Secondary Neural Cases

Cases that have previously been of the lepromatous type with polyneuritic features (mixed cases) but in which the lepromatous lesions have resolved leaving only the polyneuritic manifestations are called secondary neural



FIG 48—Advanced lepromatous leprosy.
Note inability to close eyes due to lagophthalmos



FIG 49—The all too frequent termination of advanced lepromatous leprosy—blind and mutilated a caricature of his former self but still a man with a living soul

II Special Subclassification (according to the nature of the lesions present)

(1) Lepromatous Type

No varieties of the lepromatous type of leprosy have been established that are sufficiently distinct frequent and general in occurrence to require recognition in formal classification. In some places (e.g. India) where many cases show at least for a time extensive diffuse involvement of the skin not localised in macules or infiltrations there might be an advantage in distinguishing such cases (which might be indicated by the symbol Ld) but it is not certain that this division would be generally useful

(2) Neural Type

The neural type of leprosy may be divided into two main subtypes namely anaesthetic and macular. For some purposes such subclassification may be sufficient. However for more exact work the macular variety should be divided

into simple and tuberculoid and the latter may be further divided into minor and major forms. For such work therefore the following is proposed

NEURAL (TYPE)

Anæsthetic (non macular polyneuritic) (Na)

Simple macular (with flat macules) (Ns)

Tuberculoid macular (minor and major) (Nt)

Anæsthetic This variety of neural leprosy presents evidence of involvement of nerve trunks only (polyneuritic changes and sequelæ) without macular skin lesions

Simple Macular The simple macular cases which comprise a considerable proportion of those encountered present skin lesions (leprides) that have no or only very slight elevation or palpable infiltration. When elevation is present it is often difficult to detect it in diffuse light and the surface is smooth not granular or pebbled in appearance such elevation is usually limited to a narrow marginal zone. Residual lesions which are without elevation and therefore are simple under this definition (though they may be affected by scarring) should not be considered as a separate variety

Tuberculoid Macular This subtype as stated may be divided into two groups which are here described separately

(a) *Minor Tuberculoid* The lesions so designated are the less marked ones of the kind that has become generally recognised to be histologically tuberculoid and that is clinically recognisable as such with certainty. These lesions show definite elevation of characteristic appearance though there are considerable variations. They are usually marked by irregularity of the surface due to the essentially focal nature and superficial location of the tuberculoid process. That condition may produce elevated bands or areas which may be continuous or discontinuous even to the point of producing isolated papulations. Occasionally the process is relatively deep in the dermis in which case the surface may be relatively smooth and the appearance may therefore approach that of some of the major tuberculoid lesions but the degree of the condition is less than in that form

(b) *Major Tuberculoid* The lesions so designated are the more striking grossly elevated ones to which recognition as tuberculoid has been largely confined in the past. They are major both in degree and nature of the pathological process. Typically the process invades the deeper layers of the skin to a marked degree and also the subcutaneous tissue and by further extension in the cutaneous nerves related to the macules it may produce gross involvement of them. Macules of this variety are those most liable to be mistaken for lepromata especially when they are (a) small but thick morphologically nodular (b) in a reaction condition reddish turgid and smooth or (c) bacteriologically positive. One feature that helps to differentiate them is their typically sharp demarcation and frequently asymmetrical distribution. Another occasional feature is the tendency to the development of marked enlargement of the local cutaneous nerves which condition sometimes extends to the main trunks of an affected extremity thus introducing a secondary polyneuritic element. A point of importance is the frequency with which these lesions start abruptly as a reaction condition and the relative rapidity—and sometimes the completeness—with which they may subside. (Report of International Congress held in Cairo in 1938)

Technique of Examination

It cannot be too strongly emphasised that correctly to diagnose and classify leprosy the technique of examination that one adopts must be accurate. In establishing a diagnosis and especially in determining the classification of leprosy the principles of general medicine stand. Firstly one must train one's powers of observation. Secondly one must use correct laboratory methods remembering these methods are used to confirm a diagnosis and not to make one for the diagnosis is made on clinical signs.

Firstly as regards the technique of examination always see that the patient is seen in a good light and males should as far as possible be examined with all clothes off except for a loin cloth. Women should be examined with equal care and all areas systematically seen including the buttocks.

Secondly with regard to examining for anaesthesia with care this can be done in most cases even in quite young children. In persons who are uneducated whose mental processes are slow it may not always be possible to ascertain the extent of the anaesthesia on the first occasion but with patience few will find it difficult to understand what the physician wants.

In testing for anaesthesia a feather is the most useful thing to use for it is tactile and not pressure sense that is to be tested. First demonstrate on your assistant what you want the patient to do or have your assistant test for anaesthesia on yourself. Never ask a patient whether he feels always get him to point out the place touched. If the patient fails to recognise the place go over it a second or third time to make sure that there is no mistake. Give a child frequent rests if you think the examination will be prolonged for a child gets fatigued easily. With regard to the examination of nerves never ask a patient whether a nerve is painful but look at the patient's face. A twitch of the facial muscles will indicate that the nerve is tender. Never squeeze a nerve firmly it is not necessary and if the nerve is acutely tender as it frequently is you may so frighten the patient that he will not allow you to examine him. Roll the enlarged nerve under your finger and this gentle pressure will be enough for you to determine the size of the nerve and will also elicit tenderness if present. Remember always to examine the corresponding nerve on the other side it is never safe to pronounce a nerve enlarged without comparing the one on the other side. Gross enlargement of course is obvious but it is quite striking the number of times cases which are diagnosed as leprosy with a remark that the ulnar nerve is enlarged and yet one can find no such enlargement. A good general rule is if in doubt the nerve is not enlarged. In testing for anaesthesia test systematically any visible patches on the extremities trunk and face—although as pointed out the latter area seldom loses touch sense. It may be necessary to test for heat and cold for this is not infrequently absent when tactile sense is present.

In making a diagnosis of leprosy it is essential to take skin smears to ascertain whether *M. leprae* are present in demonstrable numbers. The technique of taking smears has now become uniform the older methods of puncturing the skin with a needle and squeezing blood and serum on to a slide or by excision of a piece of tissue with a pair of scissors curved on the flat are entirely superseded by a method which has been practised in the Philippine Islands for years. The following is a description by Wade (1935) of the technique of taking smears.

(1) Cleanse the area to be examined by rubbing briefly though vigorously with a small cotton wool sponge with spirit. Wipe dry with cotton wool.

(2) Pinch up the skin in fold applying enough compression to stop or minimise bleeding. When it cannot be actually picked up compress it laterally as much as possible.

(3) With a properly cleansed scalpel of suitable style and size make a small but real cut 3 mm or so long and deep enough (about 2 mm) to get well into the infiltrated layer.

(4) If blood or lymph exudes in any quantity wipe it off. With the knife blade turned transverse to the line of cut scrape the side and bottom of the cut repeatedly and with sufficient vigour to obtain a little actual tissue pulp from below the epidermis.



FIG. 60—Pick up the skin between the fingers and exerting pressure.



FIG. 61—While still exerting pressure the skin is made to the depth.

(5) With the knife transfer the small amount of material obtained to a microscope slide and make a uniform and moderately thick smear over a small area. Multiple smears from the same patient are best put on to a single slide.

(6) For after treatment of the cut the patient is simply given a bit of cotton wool to compress it until oozing stops. No dressing is necessary.

In addition to taking a smear from lesions in the skin a nasal smear should also be taken. The technique is as follows:

In a good light a blunt but narrow bladed scalpel is introduced and the internal septum is scraped sufficiently to remove a small piece of mucous membrane and this is transferred on to a slide and teased out so that a uniform smear is obtained. It is sometimes preferable to use a speculum where there is doubt or when examination reveals a definite lesion in the nose.

The instrument we use for taking nasal smears which was suggested by Wade of the Philippine Islands is a paper clip straightened out and the end hammered sufficiently to form a small scoop. This is then fixed into a piece of wood or bamboo to act as a handle and forms a very convenient inexpensive instrument for the purpose.

It is wise so that the risk of transferring bacilli from one slide to another is minimised always to spirit and flame the knife or other instrument before taking smears from another case and if possible keep one knife and nasal scraper for the examination of neural cases and another for lepromatous. The question now arises as to how many smears should be taken from a patient. For diagnostic purposes a comparatively few need to be taken but the old advice of a nasal scraping and smears from ear lobes is not sufficient. The routine we adopt is as follows. Smears are taken from the ears forehead chin cheeks and half a dozen smears taken from suspicious lesions. When deciding to take smears always examine in a good light and take smears from the edge rather than towards the centre of the lesions. Smears from the buttocks should always be taken for as the late Dr G B Archer pointed out the skin of the buttocks is frequently positive and may remain so after all other lesions have become negative.



FIG 11 — The base of the incision is scraped still maintaining pressure

Finally before completing the description of the technique of examination it should be borne in mind that the nose is very seldom the primary source of infection. In fact it is doubtful whether a bacillus bearing lesion in the nose is ever found without corresponding lesions in the skin. In examining and staining smears it should be remembered that these should not be too thick and bits of debris from the nose or skin should be removed carefully for such material is seldom decolourised and not only makes the slide messy but may confuse the picture. As a rule it is not necessary to decolourise with acid and alcohol except in smears taken from nasal lesions for acid fast saprophytes sometimes cause great confusion. These however are not alcohol fast and therefore if in doubt decolourise with alcohol as well as acid. It will be found that in the case of acid fast saprophytes these lose their stain in the presence of alcohol. This is an important point because on more than one occasion cases have been sent with a diagnosis of leprosy on a positive nasal smear alone. With regard to staining we usually find especially when there are numerous smears to examine that it is best to immerse the slides in a glass jar for twenty minutes at room temperature rather than heat the slides. The latter method is tedious if there are many slides and difficult not to leave deposits of stain on the slide as the result of over heating.

In examining smears it is well to remember that when in doubt the organism is probably not *M. leprae*. A slide should not be declared negative unless at least 100 fields have been examined. It is useful to have some criterion as to the degree of positiveness of a slide and the following standard has been adopted at the Lady Willingdon Leprosy Sanatorium, Chingleput, Madras Presidency.

- 6+ Every field full of acid fast bacilli with many globi and intracellular forms
- 5+ Every field full of bacilli with a few globi and intracellular forms

- 4+ Bacilli in every microscopic field examined
- 3+ Bacilli are not found in every microscopic field ignoring single bacilli a group or groups of bacilli are found on an average in at least 10 per cent of the fields examined
- 2+ Ignoring single bacilli a group or groups of bacilli are found on an average in at least 4 per cent of the fields examined
- 1+ Ignoring single bacilli a group or groups of bacilli are found on an average in at least 2 per cent of the fields examined

When a smear is less than 4+ a note as to the nature of the bacilli whether they are granular beaded or breaking up should be noted. No slide should be declared positive on the presence of one bacillus. Without great experience it is extremely



FIG 63—Taking the smear from the skin to the slide



FIG 64—Arrangement of multiple smears on the slide to facilitate rapidity of examination and economy of slides

difficult to make a diagnosis on a single bacillus. When a smear is less than 1+ especially in cases where the patient was previously negative a report indicating that there are occasional bacilli seen or that a few granules or beaded forms were detected is justifiable.

The following is a brief description of the methods followed at the Lady Willingdon Leprosy Sanatorium and may be helpful in reminding readers of the technique of staining slides: Prepare the following reagent as a stock solution

- (1) *Solution A* 10 gm of basic or carbolic fuchsin in 90 cc of absolute alcohol or rectified spirit. Grind filter and keep in a separate bottle
 - (2) *Solution B* 5 cc of carbolic acid in 95 cc of distilled water. Keep this in a separate bottle
- Mix 1 cc of solution A with 9 cc of solution B. This is called the staining solution

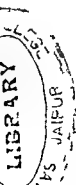
Method

(a) Pour the stain on the slide with the smears on and allow it to remain for 15-20 minutes. If there are a number of slides then immerse them in a staining jar containing the solution for this period. Wash well with running water.

(b) Decolourise by either immersing the slides in a staining jar with 15 per cent sulphuric acid for a few seconds or by pouring acid over the slide and washing with running water. The smears now regain a faint pink tint. If the colour is still distinctly red the decolouration is insufficient and acid must be reapplied. Wash well with running water.

(c) Counter stain with 10 per cent methylene blue for 3-4 minutes. Then wash and leave to dry.

The slide should now be examined with an oil immersion lens.



CHAPTER VIII

ATYPICAL LESIONS AND THE DEVELOPMENT OF LESIONS

SINCE the Cairo Conference (1938) it has increasingly been borne upon us that the classification of leprosy as set out is incomplete for it does not give recognition to what we believe are important lesions. We are of opinion that these lesions must be given a place as separate clinical entities but until this is universally recognised it is preferable to refer to them here and not incorporate them into a generally accepted classification.

1 The Preleprous Maculae or Incipient Lesion of Childhood

While this type was not included in the Cairo classification (1935) it has been recognised for some considerable time. Chiyuto and Rodriguez (1935) referred to these lesions as hazy patches. Muir (1936) refers to these lesions as juvenile leprosy and when they were first noticed by us in South India we designated them precutaneous (1937). Subsequently the term incipient lesion of childhood was coined (1941) but none of the terms are entirely suitable although we now are of opinion that the term precutaneous or what would now be called preleprous is probably the most suitable name by which to designate the lesions. The reasons for this are two fold

- (a) We believe that these lesions are true precursors of leprous leprosy
- (b) They have many of the features of leprous leprosy

The following are their main characteristics

They show themselves usually as small multiple hypopigmented patches with the appearance and distribution of leprous rather than neural leprosy. That is the lesions are slightly shiny indicating some erythema with the periphery fading imperceptibly into the surrounding normal skin so that there is no marked difference making photography of the lesions without an infra red or a deep red filter extremely difficult. There is no loss of tactile thermal or pin prick sensations. There is almost invariably a history of contact with an open case. In addition there is no nerve enlargement or anaesthesia of the extremities and the lesions are negative to standard method of examination. Their leprous is also negative. Because of their vagueness the lesions are better seen in an oblique light but bright preferably sunlight and like leprous are liable to be missed in a dull light.

Histologically the incipient lesion cannot be definitely diagnosed because there is no characteristic histological change. It consists of round celled infiltration diffusely distributed underneath the epidermis. Little evidence of focalisation and a few macrophages are usually seen here and there under the epidermis. Nerves are rarely seen in the corium and are not invaded.

It has been pointed out that this type of leprosy is comparatively uncommon as only some thirty four cases have been described in the Silver Jubilee Children's Clinic over the past five years. This represents 1 per cent of the children who have been registered at the Silver Jubilee Children's Clinic Saidapet Madras. In actual number this seems of little significance but the importance of these lesions cannot be over-estimated.

stressed 'because firstly they are difficult to diagnose and secondly a number of these cases have become lepromatous in the past five years hence the gravity of their prognosis is sufficient reason we feel for emphasising the importance of the incipient lesions of childhood

It cannot be too strongly stated that because of the difficulty of recognising these lesions in children they should not be diagnosed by the inexperienced unless they are multiple and unless there is a definite history of contact with an open case. We have already emphasised the gravity of diagnosing a case as leprosy especially in children and very great care should be exercised in the matter before the incipient or pre lepromatous lesion is diagnosed.

We are of opinion that the counterpart of these lesions is seen occasionally in adults. In one of our village surveys some years ago we came across two adults with vague hypopigmented patches with the distribution of lepromatous rather than neural leprosy but without any of the cardinal signs. Two years later both these adults on resurvey were found to have become lepromatous cases. Little is known of the existence of the prelepromatous macules in adults and therefore this is only briefly mentioned in order to draw the attention of workers to the possible existence of such a lesion.

E Atypical or Border-line Cases

Since the Cairo Conference (1938) it has been increasingly realised that the classification of neural leprosy into simple macular and tuberculoid macular does not represent the true picture neither for that matter do the two main divisions of the disease—neural and lepromatous—give a correct and comprehensive indication of the clinical aspects of leprosy. From time to time leprologists have been puzzled by certain lesions which are neither true leproma nor frank tuberculoid. Reference has been made in the chapter on Pathology to a type of tissue reaction which appears to be only partially effective and in which the cellular response of the tissue appears to be thrown into complete confusion. Until a clearer picture of this type of reaction can be obtained and we have ascertained the exact place in the classification of leprosy in which such clinical lesions can be placed it is better to refer to the whole group as atypical lesions of leprosy. The term intermediate is in some ways a more satisfactory one for both clinically and histologically these lesions represent a half way house between tuberculoid on the one hand and leproma on the other.

As has been stated this form of leprosy has puzzled workers but it has been recognised for some time particularly by Wade (1940) who uses the term border line case and Lowe (1937) who has referred to it as N^oC. South American writers speak of tuberculoid cases with negative lepromin reactions. Wade mentions some of the features of this condition particularly the more succulent nature of the lesions and the greater tendency histologically for the granuloma to be separated from the epidermis by a narrow zone which is usual in leproma. The clear zone between the epidermis and the granulomatous masses frequently shows numerous dilated capillaries. The nerves are usually grossly invaded although sometimes the invasion is less marked than in massive tuberculoid leprosy.

Clinically these lesions usually appear to behave in a manner similar to the major tuberculoid ones in that they frequently recover although the recovery period is very much longer. There are one or two curious features with regard to their progress

For one thing the patients frequently pass through a stage of extreme emaciation and one sometimes almost despairs of their lives. During the stage of activity of the lesions and emaciation the patients frequently become febrile; the fever sometimes lasting for months and not responding to the antimony products. The lesions are erythematous and percussion of the patches elicits acute tenderness. In the most severe cases they may break down rapidly and ulcerate. Altogether the patient is very miserable and causes the physician considerable concern. Bacilli can usually be demonstrated and they may be found considerably longer than in tuberculoid cases during the reacting phase—that is for more than six months and sometimes as long as eighteen months. In other words clinically these cases usually show features akin to both lepromatous and tuberculoid leprosy, but apparently they almost invariably recover. Clinically these lesions are difficult to describe, some are mistaken for tuberculoid leprosy but on closer examination the infiltration is seen to be much softer or more succulent. The latter term is hard to describe but the phrase soft infiltration as compared to hard whipcord like edges helps to give an indication of their appearance or a better idea may be conveyed by stating that these lesions look as if they contain water (Ball). Clinically the lesions appear to behave in a manner similar to tuberculoid leprosy in that they show a marked tendency to resolve although the recovery period is much longer. Frequently the lesions are much more active as seen by their spreading and less distinct edges and their greater liability to scaling and often gross ulceration. During the stage of marked activity they are usually bacteriologically positive and they remain so for nine months or more. In the ulcerative stage the patient commonly shows a marked febrile reaction (102–104° F) and this may persist as long as six or seven months. The fever is of an intermittent type with the maximum rise in the evening.

The histology of the lesions varies considerably. Some simulate tuberculoid leprosy except that there is a free sub epidermal zone which is very vascular. Others simulate leproma except that there are epithelioid cells and an occasional giant cell with the nerves in the subcutaneous tissue invaded. Still others are almost identical with the sarcoids that is masses of packed epithelioid cells with occasional giant cells. Here again a differentiating feature is the presence of gross nerve invasion. While clinically these differences cannot be diagnosed with certainty histologically atypical lesions fall into four categories

- (i) Atypical tuberculoid
- (ii) Atypical leproma
- (iii) Intermediate lesion
- (iv) Sarcoidal

Mention is made in the above divisions of the atypical lesion styled intermediate. Since the recent work of Wade (1940) Lowe (1940) and Cochrane (1940) it is evident that there is a histological picture which is betwixt and between leproma and tuberculoid and which shows the presence of giant cells and epithelioid cells with marked round celled infiltration in parts of the corium free sub epidermal vascular zone and some foamy cell change. It is these lesions with this definite histological picture to which the term intermediate is confined. It is extremely difficult to give the reader a true picture. The photographs illustrating this chapter may be of some help and the following is an attempt to summarise the main clinical and histological findings

in tuberculoid lepromatous and atypical leprosy. Photomicrographs of this condition are included in the chapter on Pathology.

TUBERCULOID

Paired erythematous and grossly infiltrated lesions with sharply defined edges which remain well demarcated even as they extend.

During reaction lesion may be tender and desquamate and occasionally ulcerate. Febrile period if any of short duration. Patient seldom needs hospitalisation. Nerve abscesses common.

Bacilli. Lesions in state of reaction positive but bacilli disappear within 3-6 months.

Histology. Granuloma extends to epidermis with no sub-epidermal clear zone. Giant cells and epithelioid cells well marked round celled infiltration concentrated around hair follicles and vessels in the corium.

Cross invasion of nerves.

Lepromin. Positive.

ATYPICAL

Raised erythematous lesions with succulent appearance the edges are infiltrated but less well defined.

During reaction marked burning and tenderness of lesions liability to ulcerate. Fever sometimes prolonged leaving the patient emaciated and extremely ill looking. Frequently requires hospitalisation. May show nerve abscesses but rare.

Lesions practically always positive and in active cases may be present for 6-9 months or more.

Granuloma respects sub-epidermal zone in which there are numerous dilated capillaries. Infiltration diffuse with less tendency for concentration of round cells. Histology varies may show lepra cells and foamy cells may show packed epithelioid masses or granuloma may consist of undifferentiated macrophages. Nerves involved frequently grossly.

Lepromin. Usually negative may be moderately positive in stage of activity.

LEPROMATOUS

Erythematous lesions in filtration usually slight if marked lesions tend to coalesce. There is no definite demarcation of edges and difficult to distinguish where lesion ends and normal skin commences for in such cases there is often no normal skin.

During reaction lesions show exacerbation of signs with rose spot nodules typical of lepra reaction. Fever with maximum rise at 4 p.m. and all signs of acute reaction (see Chapter VII). No nerve abscess enlargement of nerves due to an interstitial fibrosis.

Lesions always positive and usually take months or maybe years to become negative.

Clear sub-epidermal zone not markedly vascular. Lepra cells (macrophages). No epithelioid or giant cell. Round cell infiltration not marked but always diffusely distributed. Nerves stand out clearly with slight proliferation of perineurium. Foamy cell may be present.

Lepromin. Invariably negative.

We are of opinion that this group of atypical lesions is important firstly because of their tendency to be confused with leproma and puzzlement as to the efficacy of treatment arises as a result of their great tendency to spontaneous recovery. Secondly because they may be confused with tuberculoid lesions and hence give the impression that tuberculoid leprosy may turn into leproma. It is our opinion that tuberculoid and leproma cannot exist together for it is hardly conceivable that one part of the skin is in active and effective defence and another shows ineffective defence hence there is little justification for claiming the existence in the same section of tuberculoid

and lepromatous change. We cannot agree with Lowe (1940) that the group is not sufficiently large to interfere seriously with the Cairo Conference classification because we consider these lesions to be more frequent in South India but unless a definite note is made of them great confusion is liable to arise and workers fail to understand what appear to be anomalous phenomena. Ryrie (1934) has referred to such lesions as ulcerating tuberculoid and his description bears close resemblance to atypical lesions showing gross emaciation with marked ulceration.

Earlier workers have described a condition known as *Lazarine leprosy*. It is difficult to place this manifestation in any modern classification but it is more than probable that many cases of so called *Lazarine leprosy* may have belonged to one or other of these atypical groups which frequently shows extensive ulceration.¹

The Development of Lesions of Leprosy

There has been much speculation as to the development and method of progress of leprosy in the body. Muir (1923) visualised the extension of leprosy from an initial lesion in the skin or nerve and from this the disease spreads by an ascending infection along the nerve sheath and by the lymphatics and to a lesser extent the blood stream and thus the disease becomes generalised throughout the body. We have indicated that leprosy develops from a primary focus possibly analogous to the Ghon focus in tuberculosis and its method of spread is directly influenced by the actual or potential immunological response (tissue immunity) of the skin. When this is marked as in tuberculoid leprosy the infective agent is anchored in the skin and the disease becomes spontaneously arrested. Where it is absent there is a grave possibility of widespread invasion of the whole reticulo endothelial system and serious lepromatous leprosy results. We have suggested that the *M. leprae* finds its most favourable habitat in the reticulo endothelial system. It endeavours to invade this system while the tissues of the body whenever possible try to prevent such an infiltration either as in tuberculoid leprosy by a tissue response giving rise to an effective barrier and preventing the progress of the bacilli from the primary focus to the reticulo endothelial system or as in pure neural leprosy (neural anaesthetic) by a violent fibrous tissue response giving rise to an interstitial fibrosis. For reasons not altogether understood the *M. leprae* apparently cannot infiltrate the reticulo endothelial system except via the skin and if this is prevented no progressive leprosy is possible. This may be the explanation of the failure of animal inoculation.

If this is the case it may be profitable to discuss how far one type of leprosy is possible of developing into another type and in order to study this there must be some understanding of what are the basic lesions of leprosy. I indicated (1940) that leprosy may develop from three basic lesions—the incipient simple macule and the tuberculoid. Since writing this article Pardo Castello and Tiant (1944) have described the lesions of leprosy under non specific tuberculoid and lepromatous stating that the lepromatous and the tuberculoid types are definite clinical pathologic bacteriologic and immunologic forms of leprosy and that the non specific type represents transitional stages. These authors further give a diagram which indicates that leprosy of the skin and nerve arises from three distinct lesions and designate them lepromatous tuberculoid and non specific. Before one can accept such a scheme it is necessary to

¹ It is probable that the group of atypical lesions is ultimately passing into lepromatous and therefore the term is not final for these cases may be appropriate.

ask are such lesions the first lesions to appear? In other words does leproma commence at all times as leproma and never pass through a transitional stage and does this also apply to tuberculoid leprosy? The older conception that lepromatous lesions usually commence as neural is largely discarded

Rodriguez and Wade (1939) have made interesting and valuable contributions on the status of neural cases and the development of tuberculoid leprosy and have shown that the simple macule may develop into leproma and that the tuberculoid case may set in as a reactive phenomena in a previous simple macular case or in some instances the lesions commence as minor tuberculoid and there may be no previous simple macular phase. Over a period of seven years lesions have been observed in children in Saidapet and Tables XI and XII give to date the development of these lesions and their transformation into other types

DEVELOPMENT OF LESIONS OF LEPROSY

TABLE XI

Type	Number Registered	Number Condition not known	Number turned Intermediate	Number turned Leproma
N (ns)	238	103	1 (0.42%)	15 (7.52%)
Nt (minor)	236	—	—	3 (1.27%)
Incipient	40	3	3 (7.50%)	13 (32.50%)
Suspicious	145	48	1 (0.68%)	—

The tuberculoid minor cases that have been noted as turned leproma were clinically tuberculoid but histologically were not confirmed

CHANGES IN TYPES

TABLE XII

Type on Admission	Number Registered	Condition not known	Balance	Changed clinically to	No
N (ns)	238	103	135	Tuberculoid	29
				Leproma	15
				Intermediate	1
Incipient	40	3	37	Simple macular	11
				Tuberculoid macular (Nt)	2
				Lepromatous	13
				Intermediate	3
Suspicious	145	48	97	Simple macular	12
				Tuberculoid macular	14
				Minor	
				Intermediate	1

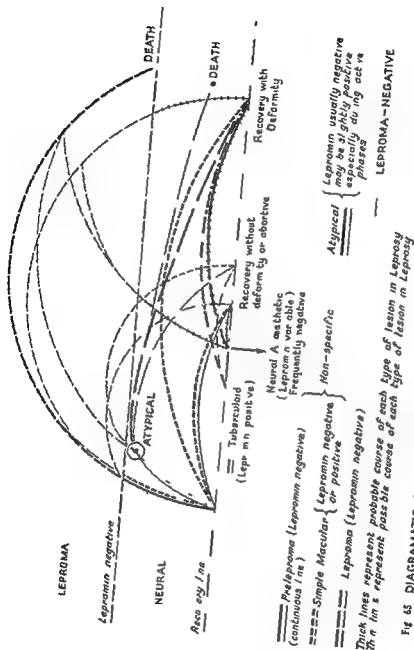


Fig 65 DIAGRAMATIC REPRESENTATION OF THE DEVELOPMENT OF LESIONS OF LEPROSY
(MODIFICATION OF MUIRS LEPROSY CURVES)



FIG 66—The prelepromatous macule or incipient lesion of childhood

deformed or burnt out case. He depicted in this representation the possibility of a few cases progressing along a low curve and reaching the advanced neural stage without passing through the lepromatous stage. Our opinion however is that in the majority of cases the various types of leprosy remain true to type that is neural remains neural and leproma continues as leproma only in the minority of cases do lepromatous lesions disappear and the patient becomes a secondary neural case. In a few instances—the number is probably higher in the early lepromas—leproma does reach the recovery line and the disease clears up without deformity. The majority of lepromas however after passing through periods of comparative or actual quiescence end in death with lepromatous signs still present. Is there then any indication which would help the clinician to estimate the likelihood of a simple macular case becoming leproma? Rodriguez and Wade (1939) indicated the greater tendency of the simple macular cases which showed multiple lesions to become lepromatous. From material in the children's clinic Saidapet Madras and the Lady Willingdon Leprosy Sanatorium Chingleput this observation is confirmed and it has also been pointed out by Cochrane Rajagopalan *et al* (1941) that approximately 48 per cent of all simple macular lesions

It has been contended by Lowe and Velasco (1941) that tuberculoid leprosy may progress into lepromatous but except in a small number of cases (1.27 per cent) in which the tuberculoid process is of a minor degree and not of sufficient intensity to react positively to leproma I have not in my experience ever been convinced that this happens. Any tuberculoid cases which have appeared to change have proved on closer study to belong to the atypical group.

The evidence of the past ten years in South India indicates that the conception that all lesions of leprosy develop from certain basic lesions appears to be confirmed and Fig. 65 is an attempt to illustrate diagrammatically the probable and possible course of development from these basic lesions viz non specific tuberculoid and neural anaesthetic. Muir (1926) visualised the course of leprosy as a series of parabolic curves passing from the neural stage through leproma and ending in the secondary neural

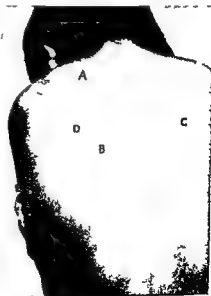


FIG 67—Note the great vagueness of the lesions, their multiplicity and distribution. They were negative to standard methods of examination.

tested were negative to lepromin. It is therefore reasonable to assume that the factors which are of greatest importance in estimating the probability of a case turning leproma are the age of the individual, the multiplicity of the lesions and the lepromin reaction. We are therefore convinced that the majority of lepromas commence from prelepromatous macules (incipient lesions in childhood, suspicious lesions in adults), some however develop from the simple macular lesions (approximately 7 per cent). There has been no evidence from the work in Madras that leproma ever starts as leproma without a previous macular stage in which it has so far been impossible to demonstrate bacilli by standard methods of examination.

It will be noted that the neural anaesthetic type has been included in the term basic lesions. We are of opinion and the reason is stated in the chapter on Pathology that leprosy may commence as a neural anaesthetic case and continue as such throughout life. Parelly however as Poy (1933) pointed out, does a neural anaesthetic case in the later stages become lepromatous.

It is impossible to place at present the atypical case in its right position and hence this is represented as possibly developing from simple macular leprosy and hence is not included in the basic lesions. It is hoped that this modification of Muir's curves in the diagram will give some appreciation of the probable course and development of the various lesions of leprosy.

NOTE.—Recently (1946) there has been considerable discussion on the classification of leprosy and the following is a modification of the Cairo classification based on the criticism of certain South American workers and as a result of the study of the disease in India, amended accordingly. This classification correlates the immunologic, histologic and clinical picture and gives, we believe, a clearer representation of the disease.

- I Non specific lesions (or uncharacteristic lesions)
 - (a) Neural macule (simple neural of the Cairo classification)
 - (b) Prelepromatous macule (incipient lesion)
 - (c) Neural anaesthetic leprosy
- II Active tissue immunity (? allergic)
 - (a) Tuberculoid leprosy (minor tuberculoid, major tuberculoid)
- III A) ent tissue immunity (? anergic)
Lepromatous leprosy
- IV Partial or varying tissue immunity
Intermediate or border line cases (atypical tuberculoid, atypical leproma and sarcoidal leprosy)

CHAPTER IX

DIFFERENTIAL DIAGNOSIS

WE are now in a position to discuss those diseases and conditions which simulate leprosy. In doing so it must be remembered that on the one hand the physician unfamiliar with leprosy seldom bears in mind the possibility of the patient having leprosy unless the disease is so obvious that it cannot be missed while on the other hand the physician who has commenced to take an interest in leprosy is very liable to diagnose everything as leprosy. A good rule for those to follow who are building up their experience is when in doubt never diagnose leprosy. Further if one refuses definitely to diagnose leprosy—at least until sufficient experience is attained—unless one or both of the cardinal signs are present a physician may miss a few early cases but he will save much distress for the fear of leprosy is liable to haunt persons in the East in the same way as the fear of cancer is in the West. Therefore always be reasonably certain before a diagnosis is made of what to many is a dreadful disease and one that is greatly feared.

It may be thought that to miss diagnosing an early lesion of leprosy will result in the withholding of adequate treatment and the condemning of the patient to life long misery through his becoming an advanced lepromatous case. It will however be apparent that this is not necessarily the case for the majority of early lesions as has been shown have a great tendency to spontaneous disappearance and as will be mentioned later the special remedies used for leprosy are not specific and therefore there is no cause to be disturbed unduly should an early case be missed and treatment not started. It is well then always to bear in mind that the two cardinal signs of leprosy are

- 1 The presence of anaesthesia
- 2 The presence of M. leprae discovered by standard methods of examination

We are of opinion that the diagnosis of the doubtful case should be left to the specialist. If the above principles were followed by the general physician in the tropics unnecessary mental suffering and useless treatment would be avoided.

In considering the differential diagnosis of leprosy those conditions which are liable to be confused with neural leprosy will first be described the differential diagnosis of lepromatous leprosy will then be discussed.

NEURAL LEPROSY

A Simple Macular

The hypopigmented lesions of simple macular leprosy have to be distinguished from the following conditions

1 Birth Marks (naevus anaemicus)

In the darkly pigmented races hypopigmented areas which appear to be congenital and possibly of a similar nature to naevus anaemicus are not uncommon. These

lesions have been mistaken for early neural leprosy. The hypopigmented areas look as if they are part of the texture of the skin and not a pathological lesion. The history will indicate that the condition has been present from birth and there will be no other signs of leprosy. An interesting observation is quoted by Sutton (1931) as to the pathology of naevus anaemicus. He states that naevi of this type are due to a congenital abnormality of the nervous apparatus of the blood vessels: the vasodilators being absent while the vasoconstrictors are present. Such an explanation may throw some light on the pathology of the hypopigmented lesion in leprosy for the reason for the loss of pigment in these lesions has never been adequately explained. In this instance the vasodilators may be affected through the nerve terminals in the skin being damaged by leprosy.

2 *Tinea Versicolor*

The distribution is different from the simple maculae of neural leprosy although when extensive they may be widespread over the body. The typical distribution is seen between scapulae front of chest face and neck. Molesworth has pointed out that in Europeans tinea versicolor practically never invades the face. It should be remembered that in Indians tinea versicolor frequently affects the face. The lesions show a fluffy appearance which has been described as the colour of coffee with milk (*café au lait*). The lesions look as if powder or fine dust has been scattered on the surface of the skin. The characteristic fungus can be demonstrated by scrapings from the scales of the skin.



FIG. 68.—Birthmark on face. Note enlargement of the great auricular nerve. Also a case of leprosy.

3 *Scars from Infected Impetiginous Lesions and Boils etc*

Wherever there has been a generalised infection with scabies and impetigo hypopigmentation is liable to result. These lesions however are small and are surrounded by a characteristic hyperpigmented border. As far as I am aware hyperpigmentation never occurs in leprosy. It should be borne in mind that impetigo in the East frequently becomes secondarily infected. Not infrequently as a result of a boil a hypopigmented patch quite large in size will develop but if a careful examination is made the scar of the depressed centre indicating where the core of the boil was will be seen. Scars of all kinds have been mistaken for leprosy and these are often anaesthetic but the atrophic condition of the skin and the absence of other signs will throw light on the diagnosis. A word of warning needs to be issued here: sometimes patients will cauterise an area in order to get rid of the signs of leprosy but a careful history will exclude this possibility. When in doubt a biopsy may help.

4 *Leucoderma*

This when well marked cannot be mistaken for leprosy but before pigment has been completely lost the lesions may show a mottled appearance and this may be

mistaken for hypopigmentation due to leprosy. The absence of cardinal signs and the distribution are all against leprosy and if the patient is watched in due course the characteristic complete loss of pigment will be noticed.

5 *Small Hypopigmentations around Mouth and Lips*

Frequently children in the tropics especially if they have a running nose develop scaly hypopigmented lesions on the cheek or in the vicinity of the lips. Similar lesions also occur in nutritional deficiencies. If it is remembered that no diagnosis should be made unless the lesion is typical a mistake will not be made. These small scaly nutri-



FIG 69 — Old chronic patch (fungus infection)



FIG 70 — Leucoderma. Note the complete vitiligo with areas of pigmentation in the centre (Photo by Dr T B V Sloan)

tional pigmentations are usually associated with angular stomatitis. No lesion on the mouth or face should be definitely diagnosed unless absolutely typical.

6 *Seborrhoea*

Extensive seborrhoea may give ground for confusion but the distribution behind the ears, axillae, under the breasts in women and around the umbilicus should help to differentiate this condition especially as it is invariably associated with dandruff in the scalp.

7 *Phrynoderma*

This condition was first described by Nicholls (1938) in Ceylon and later by Akroyd in India. It is sometimes associated with hypopigmentation. The lesion has the appearance of a lichen spinulosum: the hairs stand out from the follicles and there is no

anaesthesia. Large doses of Vitamin A should be given and this will clear up the condition if present thus establishing the diagnosis.

B Neural Tuberculoid

In considering the differential diagnosis of these lesions the following conditions must be borne in mind

1 *Tinea*

Tuberculoid leprosy is not infrequently diagnosed as a *tinea*. The fungus can be discovered by examining a scraping from the scales of the skin after soaking it in 30



FIG 6 — Extensive xeroderma of the skin

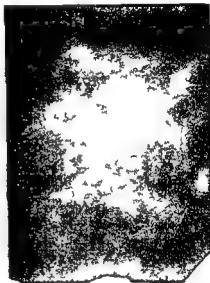


FIG 7 — A leontopetalous lesion on the abdomen (leontopetalous)

per cent KOH for at least twenty minutes or longer if necessary. When in doubt the therapeutic test can be applied for if a lesion is fungoid it will disappear with the appropriate treatment. If a lesion does not itch and has been present for 6-12 months and does not respond to ordinary medication and a biopsy should be performed it is probably leprotic in nature. This maxim is useful to remember in the case of Europeans in the tropics who may come into contact with leprosy.

2 *Syphilis*

The pleomorphic characteristics of secondary syphilis frequently give rise to great confusion. The Wassermann reaction may not help because in leprosy, especially when the lesions contain many bacilli this may be positive. The lesions of syphilis are frequently seen on the palms and soles associated with leukoplakia, a history of syphilis and finally the therapeutic test will establish the diagnosis for treatment will clear up the syphilitic lesions leaving those of leprosy little affected.

3 Psoriasis

This condition is frequently a cause of confusion for in many respects psoriasis is not unlike tuberculoid leprosy in the stage of reaction. The points of difference which should be kept in mind are

(1) The characteristic distribution of psoriasis elbows knees scalp. While we are aware that the lesions of psoriasis occur anywhere on the body lesions should always be looked for on these sites

(2) The scabiness of the lesions is very much more marked and shows itself in silver scales. On removing the scales one gradually comes down to a base in which there are small bleeding points

(3) There is no loss of sensation neither is there any enlargement of the superficial nerves

(4) It is not possible to find M. leprae in the lesions

(5) Characteristic histological changes of psoriasis and not leprosy



FIG. 73 —Secondary syphilis

4 Lupus Vulgaris

Because the histology of these lesions is similar to that of leprosy skin sections are sometimes diagnosed as skin tuberculosis or lupus when in reality they are sections from tuberculoid leprosy. It is well to bear in mind that while lupus vulgaris is seen in India and the East this is a comparatively rare condition. Whenever a section shows tuberculoid histology it is more likely to be leprosy than lupus. In lupus vulgaris the infiltration process is usually seen deeper in the corium and there is no evidence of nerve invasion. In lupus verrucosus

there are gross epidermal changes whereas in leprosy epidermal changes are seldom if ever seen. Other varieties of tuberculides such as erythema induratum papulo necrotic tuberculide are extremely uncommon and are not likely to be confused with any form of leprosy.

5 Granuloma Annulare

This again is a rare condition. I have never seen a case in India. It is mentioned because some authorities consider the condition closely related to the tuberculides. It is most commonly found on the back of the fingers its slow evolution, lack of sensory loss or nerve enlargement should serve to distinguish it from tuberculoid leprosy. Biopsy will establish the diagnosis for in granuloma annulare there is a deep seated infiltration in the corium with necrosis in the centre. In tuberculoid leprosy there is never this characteristic necrosis in the corium of the skin.

6 *Sarcoids*

The differential diagnoses of these lesions are mentioned here although the sarcoidal type of histology is most frequently seen in the atypical case. The condition is extremely rare and the Besnier Boeck syndrome is associated with lesions in the lungs, lymph nodes, liver and spleen. The histology is similar to atypical tuberculoid except that the nerves in the corium are not invaded. Pardo Castello and Tiant mention internal lesions in leprosy of the sarcoidal type but this has not been confirmed by other workers. The disease appears to be extremely rare in the tropics and is hardly likely to cause confusion. Some authorities have considered sarcoids are related in some way to leprosy but it is more likely that in these instances these lesions were confused with the atypical type of leprosy.

7 *Lupus Erythematosus*

The characteristic lesion on the nose but wing distribution with the atrophic condition of the skin should differentiate from leprosy. The histological picture is entirely different and giant cells are usually absent.

8 *Eczema*

This is only mentioned in passing for in the acute reaction phase of tuberculoid leprosy the cellular reaction may be so severe as to give rise to marked inflammation and oedema of the tissues. Especially is this the case if one side of the face only is affected. In all such cases the great auricular nerve on the affected side will be found to be enlarged the lesions show fine scaling and percussion elicit acute tenderness. Other points are absence of serous exudation and no marked itching. Usually in the acute stage of reaction M. leprae can be found by standard methods of examination.

9 *Pellagra*

This may be confused with leprosy but the distribution of the lesions in pellagra—neck, arms, exposed parts of the body—characteristically denuded and inflamed tongue accompanied by diarrhoea and possibly mental disturbances—should make it easy to differentiate from leprosy.

✓ C Differential Diagnosis of Neural Anaesthetic Leprosy

| It is frequently not realised that neural leprosy can manifest itself as a pure neural case with typical glove stocking anaesthesia and when such a case presents itself at a hospital out-patient department it is liable to be confused with the following conditions.

1 *Peripheral Neuritis*

The most common cause of peripheral neuritis is a dietetic deficiency associated with a deficiency of vitamin B complex. Less common causes are diabetes, arsenic, alcohol. If it is remembered that the reflexes are never lost in leprosy and frequently as Subramaniam has pointed out exaggerated the diagnosis of peripheral neuritis should not be difficult. In arsenical lesions of hands and particularly of the nails the presence of traces of arsenic in nails or urine will confirm the diagnosis. In diabetes the presence of sugar in the urine will help although diabetes and leprosy can occur

together. Sometimes the diagnosis can be established by cutting down on the peroneal or ulnar nerve and taking a scraping from the nerve itself after opening the sheath as the *V. lepra* can occasionally be found in the nerve sheath when it is impossible to demonstrate it elsewhere. Anaesthesia associated with nerve enlargement is always due to leprosy.

2 *Bernhardt's Syndrome*

Muir drew attention to the confusion which may arise from mistaking a neuritis of the lateral femoral cutaneous nerve for leprosy. This condition shows itself as an area of anaesthesia on the outside of the lower two thirds of thigh and is associated with subjective symptoms of numbness or formication. The etiology of the condition is obscure. Some say that it is due to a fibrositis of the muscles and the nerve symptoms are secondarily due to the constriction of the nerve fibres in fibrous tissue and thus giving rise to anaesthesia. This condition may be similar to that described by Sutton (1931) who attributes the first description to T. C. White and names it meralgia paresthetica and attributes the cause to a defective arch which results in changes in the foot or in the comparative position of the long bones. Any cases which I have seen have occurred in adults with definite foci of sepsis in the teeth. The limited distribution of the anaesthesia, the history and age of the individual should serve to avoid confusion.

3 *Syringomyelia*

This is a rare disease and should not be diagnosed without unequivocal evidence. Dissociated anaesthesia is present i.e. loss of thermal and pain sensation. This takes place before tactile impairment. The upper limbs are usually affected and the lower seldom. Muscle atrophy is seen before sensory loss and deformity and scars from burns are common signs. There is no enlargement or tenderness of superficial nerves.

4 *Cervical Rib*

This affects the upper part of the extremity usually on one side only. The patient complains of heaviness of limb. The stimulation of sympathetic on affected side is some times noted and the subclavian artery may be more prominent. A ray will reveal the condition.

5 *Raynaud's Disease*

This is a very painful complaint with signs of vasomotor disturbances. The disease starts at the tip of the fingers with anaesthesia of the affected part only. There is no enlargement or tenderness of nerves. The pain of the lesions should differentiate it from leprosy.

6 *Trophic Ulceration*

Ulceration due to syphilis and diabetes should be kept in mind but these do not usually show marked bone involvement and are liable not to be so extensive as in leprosy. In diabetes sugar will be found in the urine. In syphilis there is strongly positive Wassermann but it must be borne in mind that both conditions may complicate leprosy. The knee jerks however are not absent in leprosy as they are in neurosyphilis and there will be other signs of tabes dorsalis.

7 Facial Paralysis

It must be remembered that Bell's paralysis is not uncommon in India as well as such conditions as syphilitic invasion of the facial nerve and facial paralysis may also be the result of a cerebral lesion. Facial paralysis without other signs of leprosy must be very rare and a diagnosis of leprosy on this sign alone should not be made.

Other Conditions

The following conditions may be mentioned but are too rare or present so little difficulty that description is not necessary. (a) Scars following burns. (b) Yaws. Scarring due to yaws may occasionally cause confusion. (c) Dejerine de Sotot's syndrome. This is a condition which shows enlargement of nerves associated with talipes and other congenital deformities and is very uncommon.

Injury and Tumour of Nerves

It must be remembered that an injury will not only give rise to permanent damage to a nerve but may be the exciting cause of a peripheral neuritis. In cases where there is a definite history of injury unless the diagnosis is unequivocal great hesitancy should be shown before a diagnosis of leprosy is made. The surest method of differentiating a tumour of the nerve from leprosy is by operating and seeing the condition of the nerve. In nerve abscess the whole nerve will be thickened with more marked swelling at the place of the abscess. Neuro fibromata are seen in isolated nodules in the nerve without corresponding enlargement of the whole nerve.

D Differential Diagnosis of Lepromatous Leprosy

If it is borne in mind that in all cases of active lepromatous leprosy *M. leprae* can be demonstrated by ordinary standard methods of examination mistakes will seldom be made by the physician who exercises care. The following conditions have to be remembered.

1 Dermal Leishmaniasis

This condition simulates lepromatous leprosy so closely that unless careful bacteriological examinations are done the patient's condition will be wrongly diagnosed. Suspicion is aroused as the distribution of lesions are not typical. There are small hypopigmented lesions most marked in the axillary region and back of the neck associated with the infiltrated erythematous lesions also nodules closely resembling those of leprosy. Usually there is a history of kala azar which has been insufficiently treated. Leishman Donovan bodies are difficult to find but if not discovered in the skin may be demonstrated in a smear from a sternal puncture. The consistent absence of *M. leprae* however should put the physician on his guard.

2 Syphilis

As has been mentioned the Wassermann reaction is frequently positive in lepromatous leprosy and cannot be relied on. Absence of bacilli distribution and history help to confirm the diagnosis.

Nodular leprosy if bacteriological examinations are properly carried out should



FIG 74 —Dermal leishmaniasis showing typical hypopigmented lesions —



FIG 75 —Dermal leishmaniasis showing hypopigmented lesions infiltrated lesions and nodules.



FIG 76 —Nodules of dermal leishmaniasis



FIG 77 —Lesions of the face simulating leprosy (dermal leishmaniasis)

be easy of detection. The following conditions have at various times been confused with the disease : adenoma sebaceum leukaemia acne erythema nodosum. A word might be added on the last two conditions : the pitted infiltrated and somewhat oedematous face may make one suspicious but the presence of comedones and the absence of M. leprae establishes the diagnosis. The only reason why erythema nodosum is mentioned is that this condition can very closely simulate the rose spot nodule of leprae reaction. The absence of bacilli however would raise suspicion that the diagnosis is wrong. It should be remembered that in the fair Indian and European lesions of leprosy sometimes have a purplish hue. any such lesions which have been present for more than six months should excite suspicion and smears should be taken. Careful examination will result in the establishment of a diagnosis.

II *Lichen Planus*

This is only mentioned in passing for occasionally the infiltrations of lepromatous leprosy show themselves as small flat topped papules. The distribution of the lesions their intensive itching and in the fair person the characteristic bluish or lilac tint of the lesions are points worthy of notice. Bacteriological examination will settle the diagnosis.



CHAPTER X

REACTION IN LEPROSY

MUCH has been written about reaction in leprosy but for this condition to be properly understood it is necessary to discuss the subject under two heads

- 1 Signs of activity
- 2 Signs of reaction
 - (a) In neural cases
 - (b) In lepromatous cases (lepra reaction)

These conditions are separately discussed for it is to be remembered that leprosy in many instances is an actively progressive disease and until the disease becomes quiescent either spontaneously or as a result of treatment the patient has to be considered as an active case. During the period in which he is active however there is a great tendency greater in some types than in others for acute exacerbations of the condition to arise from time to time and it is these more acute phases that come under the term of reaction. If this then be the case it is of utmost importance to be able to judge whether a case is active quiescent or arrested quite apart from the fact whether the activity of the case is such that it must be concluded that the patient is in a reactive condition. The following therefore are the signs that the disease is still active

- 1 Increase or decrease of lesions or anaesthesia
- 2 Erythema and infiltration
- 3 Tenderness of nerves
- 4 Presence of bacilli

One or more of the above signs may be present without the patient actually being in a state of reaction. For instance the hypopigmentation of a simple macular lesion may extend or the anaesthesia of a limb may increase so long as there are changes in the colour erythema or the extent of the anaesthesia the disease is active. On the one hand the lesions or anaesthesia may show gradual extension and the disease progress whereas on the other signs may diminish and the disease gradually retrogresses. No lesion can be considered quiescent so long as there is any erythema or residual infiltration. I am well aware as has already been indicated that many neural lesions especially of the minor tuberculoid type spontaneously retrogress but until all infiltration disappears and only a scar remains the lesions must be considered progressive. Whether active measures in the way of treatment or precautions against infecting others have to be taken is entirely another matter and these questions are discussed elsewhere. All that is emphasised here is that so long as the above signs are present leprosy whatever the type must be considered to be active either actively progressing or actively retrogressing. It must be noted that enlargement of the nerves is not mentioned as a sign of activity but only tenderness. While generally speaking a large nerve is also tender much of the enlargement may be due to an interstitial fibrosis and the nerve tissue be mainly replaced by fibrous tissue and therefore mere enlargement of a nerve is not necessarily a sign of activity of the disease. To summarise

therefore it might be well to give the definition of active cases as laid down at the Manila Conference (1931) held under the auspices of the Leonard Wood Memorial Fund (now American Leprosy Foundation). This conference defined active cases as follows: Active cases are those in which there are clinical or microscopic evidences of progressive or recessive changes in lesions with or without accompanying systemic disturbances. These evidences include the following: positive bacteriological findings in skin or mucous membrane determined by the usual method; the presence of raised erythematous lesions; increase or diminution of lesions in size or number; tenderness of nerves with or without thickening.

In connection with the consideration of active cases the question arises as to when a case ceases to be active. A case may be active but non infective and therefore it is apparent that the following states need defining:

- (a) A non infective case
- (b) A quiescent case
- (c) An arrested case

Non infective Case

Non infective cases are those which have been negative to standard methods of examination for three months and have shown no signs of activity for a similar period. Examinations being made each month during this period. The examination should include a clinical examination and a microscopic one from the nasal mucosa and from lesions of the skin from at least eight sites.

Quiescent Case

Quiescent cases are those which have been negative to standard methods of examination for six months and have shown no signs of activity for a similar period. Examinations should be made each month during this period and should include a clinical examination and a microscopic one from the nasal mucosa and from lesions of the skin from at least eight sites.

Arrested Case

Arrested cases are those which have remained quiescent for two years. Examinations should be repeated every three months if possible and not less frequently than every six months.

It is unwise to encourage lepromatous cases to cease treatment until they have qualified for a quiescent certificate.

I am aware that in defining these terms the Manila Conference recommendations are not rigidly followed in the definition of a quiescent case; the reason for this is because it is thought that the standards laid down at the Manila Conference are not sufficiently stringent.

We are now in the position of being able to discuss the general subject of reaction in leprosy. Reaction in leprosy either shows itself as an acute response on the part of the tissue to the invasion and/or multiplication of the bacilli or else as the result of the breaking down of leprotic foci. The former condition is seen almost solely in neural cases whereas the latter is only seen in lepromatous cases. It is extremely important to differentiate between these conditions for in the failure to recognise these two types

of reaction lies the chief reason why there seems to be so much confusion in regard to this subject

Reaction in Neural Cases

There are no signs of acute reaction in the simple macular or neural anaesthetic type of neural leprosy probably because the bacilli are relatively scanty and therefore no marked tissue response is seen. These cases are judged on the question as to whether they are active, quiescent or arrested and according to the definition just given.

It is true however to say that simple macular cases may pass into a state of reaction or bacilli may appear in the lesions when this happens the case no longer remains a simple macular one but has probably passed on the one hand from the simple macular stage to the tuberculoid or more rarely intermediate and on the other hand has become leproma. Therefore the description of reaction in neural cases is solely confined to tuberculoid macular cases or to the atypical intermediate or border line type the latter can be considered to be neural for nerve involvement is a prominent sign.

Reaction in tuberculoid cases is a manifestation of an acute tissue response and therefore all cases which show a positive lepromin (potential tissue immunity) are liable to react violently at any attempt on the part of the bacilli to multiply and endeavour to extend from the skin and nerves to the systemic system for as has already been explained it is via the skin or nerves that the bacilli reach the reticulo endothelial system and the case becomes an active leproma. This transformation is not possible in tuberculoid leprosy because of the marked and frequently sudden tissue response anchoring the infective process in the skin and preventing the bacilli from parasitising the reticulo endothelial system. What presses the trigger so to speak and ushers in this acute reaction condition it is impossible to say, possibly for some reason e.g. injudicious treatment indigenous remedies or the nature of the disease the bacilli start multiplying rapidly but unlike true lepra reaction there is an immediate response on the part of the cells and all the signs of active defence are seen. The main signs are as follows

- 1 Exacerbation of lesions which become erythematous with thickened raised and well marked edges. In the more acute forms the lesions will become scaly and may actually ulcerate.

- 2 Associated with this the lesions may become oedematous or the oedema may show itself apart from any lesions although other lesions will be affected as well the oedema may simulate angioneurotic oedema in the lips or the loose tissue under the eyelid may become swollen overnight.

- 3 The lesions are hypersensitive and in the more acute stages the patches may be exceptionally tender percussion on the patch eliciting agonising pain due to extreme sensitiveness of all nerve endings to the affected skin. This results in an intense burning and pricking. The lesions are sometimes so acutely tender that even the gentlest stroking causes acute pain.

- 4 Under such circumstances it is to be expected that the cutaneous nerves are in the same condition and will be found to be thickened swollen and acutely tender.

- 5 Associated with this condition one frequently finds the extremities especially the feet and hands swollen and oedematous pitting on pressure.

- 6 The same conditions may affect the nasal mucosa and distress is caused by swelling and blockage of the nasal passages.



FIG 78
Major tuberculous leprosy showing moderate tissue reaction



FIG 79



FIG 80



FIG 81

Same case five months later. Lesions completely subsided. No treatment of any kind given.

In the reaction of true major tuberculoid leprosy the breaking down of the lesions usually falls short of extensive ulceration. In the border line case this tissue reaction frequently goes on to gross ulceration and as a result the patient may run a continuous fever, become markedly emaciated and if not cared for may die. While the reaction condition lasts much longer in the border line case recovery is frequently just as dramatic although the scarring effect of the reaction is more pronounced. In a few cases the result of the reaction is seen in the breaking down of the tissue response and the patient gradually passes into the lepromatous type. In tuberculoid leprosy the reaction may be very violent but seldom lasts more than three months whereas in the border line case the condition may persist for six or seven months. Again in tuberculoid cases apart from nerve damage through intense tissue reaction in the nerves going on to abscess formation and possibly later to fibrosis reaction is always beneficial and the more acute the tissue response the better the prognosis and the more rapid the subsidence of the reaction. In the atypical cases however acute reaction while it may dramatically subside has to be viewed in a much more guarded way for actual tissue immunity as seen in the lepromin reaction may be absent or slight and therefore there is always the risk that the reaction may result in the case afterwards becoming lepromatous through the breaking down of this partial tissue immunity.¹

Reaction in Lepromatous Cases

True lepra reaction only occurs in the lepromatous case and is an entirely different phenomenon. It can best be defined as a state of the body which is produced by the breaking down of leprotic foci. Some authorities explain the fever on a toxic basis others suggest that it is an allergic phenomenon. If one were to accept the former theory that lepra reaction is a toxic process one would have to assume that toxins are liberated suddenly for in the non reacting phase where bacilli may be just as numerous there is no fever. The toxic theory seems only acceptable on the hypothesis that the sudden multiplication of the *M. leprae* and the breaking down of the bacilli results in excessive liberation of protein and other bacterial metabolites and thus produces a fever similar to protein shock. We cannot recommend this theory for we cannot visualise an allergic process without signs of allergy. In lepra reaction the lepromin test—the only known test of tissue immunity—is invariably negative. Further there is no evidence histologically that there is any tissue immunity. In Chapter IV it has been suggested that lepra reaction results from the intensive mobilisation of macrophages in their attempt to dispose of the bacilli which have suddenly multiplied. This results in an excessive stimulation of the reticulo endothelial system upsetting the fine equilibrium between the parasite (i.e. the *M. leprae*) and its host and setting up a febrile condition which continues so long as this equilibrium is disturbed. This explanation brings the reaction into the realm of defence. Lepra reaction appears to be a defensive process but one which is unsuccessful and always leads to a further dissemination of the organism and therefore harmful to the patient. Lepra reaction is seen in lepromatous cases both treated and untreated. If leprosy advances sooner or later bouts of fever are experienced and the patient passes through periodic attacks of lepra reaction. Under careful treatment the incidence of acute lepra reaction can be reduced considerably. Certain individuals especially in the early stages if carefully

Further experience leads us to believe that the vast majority of these cases pass into leproma. The change from intermediate to leproma is frequently seen on their first relapse.



FIG 83—Atypical nasopharyngeal carcinoma (immunology)



FIG 84—Nine months later



FIG 85—Atypical nasopharyngeal carcinoma (immunology). Note edema of the subglottic area (no reaction to immunology)



FIG 86—Four months later

treated never have reactions but other more advanced cases pass from one reaction to another until death intervenes from some intercurrent disease or from the cachexia resulting from prolonged fever

Lepra reaction can be divided into acute subacute or chronic and the following are the main signs and symptoms of the condition

- 1 Fever
- 2 Rose spot nodules or evanescent erythematous rashes
- 3 Increase of lesions and exacerbation of existing lesions
- 4 Subcutaneous nodules
- 5 Nerve pain and tenderness (associated with joint and bone pain)
- 6 Iritis orchitis and lymphadenitis
- 7 Breaking down and ulceration of nodules
- 8 Swelling and oedema of feet and hands
- 9 Acute laryngeal symptoms
- 10 Multiplication of the bacilli as shown by deterioration of smears (This is sometimes rapid)
- 11 Sedimentation index test raised

Emphasis must first be laid on acute lepra reaction which may be ushered in by a feeling of malaise and even if patients do not at first feel unwell when the temperature is established they usually complain of discomfort headache and body pain The temperature is characteristic there is always an evening rise and the maximum is usually seen between 3 p.m. and 5 p.m. but the highest peak may be reached by 4 p.m. In the morning the temperature is frequently subnormal The onset of the fever in the evening is not infrequently accompanied by a slight rigor This may be marked if the temperature rises above 102° F The following are the more important signs (apart from fever) which are associated with this condition

- 1 Exacerbation of existing lesions or eruption of fresh lesions
- 2 Rose spot nodules and/or evanescent erythematous rashes
- 3 Subcutaneous nodules
- 4 Iritis neuritis and occasionally orchitis

We feel that the diagnosis of acute lepra reaction is not justified unless there is fever and one or more of the above signs present In fact the cardinal signs of this syndrome are

- (a) Fever of characteristic type
- (b) Exacerbation of existing lesions or eruption of fresh lesions
- (c) Rose spot nodules or in fair persons evanescent rashes of an erythematous nature

Fever of Characteristic Type

The fever of acute lepra reaction is characteristic If a patient who is going to develop reaction is watched indications of the onset of fever can be seen in the temperature chart for there frequently develops what might be called a pyrexia below the normal line Everyone shows regular variations of temperature between the early morning temperature on rising and the evening This variation is steady but in a patient before the onset of reaction a greater degree of swing is noted below the normal line and on the onset of the reaction the temperature rises above 98.4° F A characteristic of the fever is that it is a hectic type of temperature subnormal in the morning



FIG 86

Major tuberculo d leprosy—acute & extensive stage. This type of reaction is unusual in typical tuberculo d (photograph taken 3rd December 1941)



FIG 87



FIG 88

Lesion ulcerated for some months. Lesion protected by oil—patient kept in bed and a er die. Unless given careful nursing care might succumb from a secondary infection (photograph taken 30th November 1941)



FIG 89

and high in the evening where it is possible to take regular twice daily temperatures it is wise to ask the patient always to take his evening temperature between 3 and 5 p.m. The matter will be further discussed under the control of treatment. Another point of some importance is that any departure from the usual swinging temperature of lepra reaction should be noted for a continuous fever or a remittent fever instead of an intermittent one may be the first indication that some complication has set in. While when the level of the fever reaches the higher peaks of 102–104 the patient may feel ill it is remarkable how frequently he is hardly conscious of fever and is apt to remark on inquiry that he feels quite well.

Exacerbation of Existing Lesions or Eruption of Fresh Lesions

In any patient with lepromatous leprosy the first sign of reaction may be slightly increased erythema of the lesions. This is particularly noticeable in the fair skin. With this increase in the activity of the lesions themselves the eruption of fresh lesions is frequently noted and quite apart from the rose spot nodules which are about to be described any deterioration of the patient's condition either clinically or bacteriologically should be a warning sign to the physician who should keep the patient under careful observation. It should be remembered that if reactions are to be prevented all slight variations in the temperature general and clinical conditions of the patient should be carefully noted. One word might be said with regard to the sedimentation index test. While it is true that a rise in the sedimentation index may be an indication of the onset of reaction the physician should not rely on this test overmuch but if he employs it he should correlate it with the temperature clinical conditions and bacteriological findings in order to obtain a complete picture so that he can the more accurately estimate whether the patient is passing into lepra reaction. Reliance solely on the sedimentation index test is liable to lead the clinician astray. I have seen a patient in whom other signs indicated the imminent onset of reaction with a sedimentation index of eight pass into acute lepra reaction and within twenty-four hours the whole body was covered with so many rose spots that they were separated from each other by less than half an inch.

Rose-spot Nodules or in Fair Persons Evanescent Rash of an Erythematous Nature

The rose spot nodule is typical and varies from the size of a pea to lesions which may simulate erythema nodosum. The rose spots are painful on pressure and are usually found on the extensor aspect of the limbs back and face. The occurrence of the lesions on the face and back serve to differentiate the condition from erythema nodosum. We have seen a case in which the whole body was covered with rose spot nodules. Each individual lesion seldom persists for more than twenty-four hours but they may appear in successive crops while acute lepra reaction lasts. Along with the rose spots or apart from them in the fair person vague erythematous rashes may be seen they come and go and may cause great confusion if not recognised. Some writers have referred to these as prodromata of leprosy but I believe that all so-called prodromal lesions are indications of reaction in which the early signs of lepromatous leprosy have been overlooked.

Occasionally accompanying the signs of reaction oedema of the hands or feet is seen. This oedema resolves on the subsidence of the reaction.

A warning which we feel is essential is that fever and leprosy do not necessarily

mean lepra reaction It is sometimes advisable to withhold any special treatment for reaction for three or four days so as to enable a thorough physical and if necessary laboratory examination to be completed before a definite diagnosis of lepra reaction is made. Such conditions as pleurisy pneumonia tuberculosis typhoid kala azar are all liable to be overlooked if the physician's first thought is lepra reaction when a patient with leprosy suddenly becomes febrile.

Subacute Reaction

Acute lepra reaction usually responds to treatment but if a patient has many attacks of acute reaction he is liable to pass into the subacute or chronic reaction. In subacute reaction there is a progressive increase of the disease with periodic bouts of fever which may last for a few days or a week or more followed by a period of low fever and accompanied by signs of reaction. The following signs are more prominent in the subacute phase

- (a) Continuous appearance of fresh lesions
- (b) Subcutaneous nodules are much more marked and tend to persist
- (c) Rose spot nodules continue to come and go although the subcutaneous nodule is more characteristic of subacute reaction
- (d) Iritis lymphadenitis orchitis
- (e) Neuritis is sometimes a prominent symptom and may give rise to much distress
- (f) Swelling of the hands or feet or both may be seen and results in permanent damage to the tissues due to the trauma induced from venous and lymph stasis

Chronic Reaction

This condition is usually seen in the advanced lepromatous case. The patient has considerable discomfort and usually runs a low fever which from time to time and for short periods may be high. Subcutaneous nodules bone pain and joint pain are often marked and frequently bone pain is very severe suggesting a chronic osteomyelitis. In chronic reaction subcutaneous nodules sometimes as large as a pigeon's egg tend to become fixed to the skin and break down and ulcerate. This ulceration may be very extensive and cause acute distress. Again if the hands are affected permanent damage due to venous stasis may result ending in an unsightly enlarged and deformed hand or foot. In addition lesions of the nose and throat and mouth may show signs of activity and give rise to a painful condition of the mouth and throat. In the throat signs of obstruction of the trachea may be present and if oedema of the glottis sets in may necessitate a tracheotomy. Besides this severe iritis may occur and the eye condition shows rapid and marked deterioration. Along with the distressing symptoms there is frequently severe nerve and bone pain. The lymph nodes when enlarged tend to break down and end in extensive ulceration. frequently the enlargement and suppuration is due to a concomitant tuberculosis infection for sections often reveal histological evidence of tuberculosis. The patient becomes very miserable and the condition may last for months leaving him emaciated and unable to resist intercurrent infections—e.g. tuberculosis bowel or kidney disease—which may be the direct cause of death. In all the long history of leprosy there is no more pathetic figure than the patient with chronic lepra reaction. He lies out for death and death does not come.

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Some authorities consider that lepra reaction is beneficial but this is never the case. A short sharp reaction readily controlled may do little harm but more frequently the patient's condition deteriorates and after each reaction it is more difficult to bring the body into a state of equilibrium.

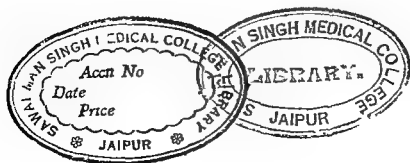
It will now be realised how important it is to differentiate between lepra reaction and reaction in the tuberculoid case. The former is ineffective defence in its worse phase the latter is active effective defence the former is invariably harmful the latter apart from the nerve damage which may result is favourable. In reaction in tuberculoid leprosy generally speaking the more acute and alarming the signs the more rapid is the recovery which sometimes is so remarkable that the patient's friends may attribute miraculous powers to any remedy which may by chance have been used at the time of reaction. Generally speaking this description also applies to reaction in the atypical case the difference being that whereas reaction in the tuberculoid case is measured in days or weeks in the atypical or border line case it continues for months frequently six or more.

If these two separate and distinct phenomena are not carefully differentiated great confusion

and not a little dismay may result. For instance if the physician mistakes acute lepra reaction for reaction in the tuberculoid case or in the intermediate case a relatively good prognosis may be given to the dismay and disappointment of the patient with subsequent loss of confidence in the physician.



FIG 90 — Enlarged deformed hand amputated because deformity and oedema rendered it heavy useless and unsightly (Incisions in fingers represent places where tissue was taken for biopsy after amputation)



CHAPTER VI

LESIONS OF THE NOSE EAR MOUTH AND THROAT LESIONS OF THE EYE

1 Lesions of the Nose

The importance of nasal lesions in leprosy has been stressed by many writers of this and last century. The opinion however that the nasal mucous membrane is the first site of infection we believe cannot be accepted. It is our belief that an initial lesion in the nose does not exist and therefore examination of the nasal secretions for *M. leprae* as a diagnostic test for leprosy is not only unreliable but futile in early cases. I personally have never seen a primary nasal lesion. Cases have been sent to me as such but either the *M. leprae* were found elsewhere or the bacilli found in the nose were not *M. leprae* but one of the many acid fast saprophytes which abound in that region. If the nasal mucosa is found to be positive without the discovery of *M. leprae* somewhere in the skin then it is wise to repeat the examination of the patient carefully and to re-examine the nose and be certain that the bacilli found are alcohol as well as acid fast and to be quite sure that no skin lesions have been missed. The only condition in which the nasal mucous membrane will be found positive without corresponding skin lesions is in the exceptional case which has lost signs of the skin infection and the nasal lesion is a residual one. While a nasal examination for *M. leprae* is useless in the confirmation of a diagnosis or making a diagnosis in leprosy the examination of the nose and the subsequent treatment of nasal lesions is of utmost importance. Apart from the added feeling of well being clean nasal passages give to a patient a person with positive nasal secretions is relatively more infective than one in whom the bacilli is only found in the deeper parts of the corium of the skin.

In examining the nose the technique is important. Swabbing the nose with cotton wool is a most unreliable method. It has been shown by Prabhu that by means of a swab of the nasal mucous membrane a nose may be completely negative but when a small piece of mucous membrane is removed examination may reveal that it can be as much as five plus positive. Hence the great importance of taking a careful scraping from the nose. A sharp instrument is not recommended, a small scoop such as described in the chapter on the technique of examination or a small Volkmann's spoon should be used. The nose should be seen in a good light preferably with a nasal speculum and the internal septum on both sides gently scraped. Each side is scraped separately and smears of each made on a slide. In addition any suspicious area of inflammation or ulceration in the posterior part of the nose should be similarly scraped. If such nasal smears are decolourised with alcohol as well as acid the error of mistaking an acid fast saprophyte for *M. leprae* will largely be avoided.

Nasal lesions in leprosy can be divided into those seen in neural leprosy and those in lepromatous leprosy.

(1) Nasal Lesions in Neural Leprosy

These only occur in the tuberculous case especially in the phase of reaction. It has been pointed out previously that where there are signs of tissue immunity any

part of the body in which the bacilli are active and multiply will show the same essential response. The nasal mucosa is no exception. In tuberculoid leprosy associated with lesions of the face the nasal mucous membrane particularly over the turbinate bones becomes oedematous and inflamed causing in some cases acute distress. These signs disappear on the subsidence of the reaction phase as a rule there is no marked ulceration although on account of the blockage and the holding up of secretions with the irritation involved superficial ulceration may be seen. This however does not give rise to permanent damage for as soon as the cause of the blockage is removed on the subsidence of the reaction the nasal passages become free the ulceration heals and the mucous membrane returns to normal. In the acute phase recovery is speedy and dramatic in the more chronic type of reaction when a state of activity persists perhaps for several weeks or more the mucous membrane tends if vigorous treatment is not applied to continue in a state of turgescence with consequent discomfort to the patient. So long as the tuberculoid condition is active just so long will *M. leprae* be found in smears taken from the nose.

In lesions of the intermediate border line or atypical variety the nose is frequently positive due to the process extending to the nasal mucosa. The condition is similar to that seen in tuberculoid leprosy bearing in mind that the duration is more prolonged and therefore positive nasal smears are liable to persist for a longer period than in tuberculoid leprosy. In both the tuberculoid and atypical forms the inflammatory process may extend to the lachrymal apparatus and the lachrymal gland become involved.

(b) *Lepromatous Leprosy*

At any stage in lepromatous leprosy *M. leprae* may appear in the nose. As already stated it is our belief that the nasal mucosa is never the seat of the initial or primary lesion. The spread to the nose is probably via the lymphatics or more rarely and in the reaction phase through the blood stream. It is to be expected that as leprosy advances so the incidence of nasal lesions increase. From an analysis of over six hundred cases at the Lady Willingdon Leprosy Sanatorium Roy gives the following percentage with discoverable *M. leprae* in nasal smears.

Type	Positive Nasal Finding	Negative to <i>M. leprae</i>
L1	36.09%	63.41%
L2	85.88%	14.12%
L3	100.00%	nil

The early changes in the nose have never been thoroughly investigated but to start with there seems to be little pathological change. The mucous membrane is slightly inflamed secretions more active and the patient may appear to have a cold although *M. leprae* may be discovered in nasal smears with little or no clinical involvement. Usually however the mucous membrane shows its state of activity by bleeding easily. At other times in addition to the inflammatory condition of the mucous membrane small greyish white or yellowish spots appear on the mucous membrane of the internal septum. As the process extends small punctate ulcers tend to develop. These are seen particularly on the septum and may result in a sharp epistaxis if they erode into a small vessel.

In all these conditions during the active stage lepromatous ulceration with granulomatous tissue is seen. On healing much scarring results and thus leads in a



FIG 91—Atypical border line or intermediate lesion. Not seen in to is condition of face and nose. Histologically how did the foamy giant cells and foamy cell with few subperidural vacuoles (see Fig 8)



FIG 92—Eight months later and tumor completely cleared. No facial features not lost



FIG 93—A ulnar type of lesion of the intermediate border line as seen in the present study. The lesion is



FIG 94—Seven months later. Two years later the boy relapsed and he now looks more like a person. No further biopsy possible

number of cases to atresia of either one or both nasal passages. It can be readily realised that in such a chronic inflammatory process almost any pathological condition may be seen and deformity of the nose is frequently marked. In passing it should be noticed that the depressed nose in leprosy differs from that in syphilis in that in the former it is the nasal cartilages that become eroded whereas in syphilis the nasal bones are affected. While the small bones in the nasal cavity become secondarily affected through spread of the infective process resulting in necrosis the nasal bones never become involved in the same way as syphilis therefore the characteristic deformity of syphilis is absent.

The following table gives an analysis by Roy of over 600 cases and indicates the percentage of the varying types of lesions seen in the nose in lepromatous leprosy.

TABLE VIII

Type	Mucous membrane inflamed	Yellow spot	Ulceration (tuberculate)	Ulceration (epithum)	Hæmorrhagic spots	Crusts and ulceration	Perforation (nose)	Atresia (nasal passage)	Occlusion (nasal passage)	Extensive to tertiary ulceration	Depressed bridge (nose)
L1	10.7	8.6	6.1	11.29	18.30	5.8	—	3.66	—	1.22	1.53
L2	11.15	7.52	1.91	22.3	4.96	31.17	2.67	7.52	1.91	6.11	22.14
L3	—	—	—	40.0	—	60.0	8.0	32.0	28.0	28.0	40.0

In the lepromatous case with extensive nasal involvement the oedema of the mucous membrane with encrustation and the consequent blockage results in a foul smelling discharge from the nose giving rise to the heavy and musty odour so often associated with advanced lepromatous leprosy. Much relief from these distressing symptoms can be achieved by systematic and careful treatment.

2 Lesions of the Ear

It is interesting to note that although the nose is so extensively involved and in spite of the close connection of the nose through the Eustachian tube with the internal and middle ear there has never been described nor have we ever seen lesions of the internal auditory apparatus. Lesions of the ear are entirely confined to the pinna. Ear lobes are affected in tuberculoid leprosy and in the atypical forms and in lepromatous leprosy. In tuberculoid leprosy and in the atypical forms the lesions are usually unilateral or much more pronounced on one side than on the other and associated with lesions of the ear lobes will be found enlargement sometimes gross of the auricular nerve on the affected side. In the atypical forms the lesions very closely simulate leproma and unless the patient is examined thoroughly the clinician is liable to err in diagnosing the type of the disease for while frequently lesions on the body will show atypical features the atypical characteristics are not so marked on the ears.

Lesions of the ear in lepromatous leprosy may vary from hardly noticeable infiltration or erythema to gross thickening or nodules on the ear lobes. The earliest site for



FIG 9

Tuberculous lipomy affecting the left ear—note swollen appearance—whereas the right ear is unaffected



FIG 9a



FIG 10 —Deep infiltration of earlobe



FIG 10a —Earlobe lightly involved

nodules to appear in lepromatous leprosy are the ear lobes and frequently on the posterior aspect. Hence the importance of seeing that the patient stands with his back facing the examiner. It is worth while to remember that ear lobes may be the first site which shows the presence of *M. leprae* and therefore no examination can be considered complete without having taken careful smears from both ear lobes.



FIG 99—Moderate leprosy with infiltrated ear lobes



FIG 100—Note nodules on the posterior aspect of pinna

3 Lesions of the Mouth and Throat

It is to be expected that when the *M. leprae* invade so extensively the skin of the face and especially when the nose is grossly involved that the mouth and the naso-pharynx do not escape. In all probability lesions of the mouth nasopharynx and trachea arise by direct spread. Although extension via the blood stream cannot be excluded it is interesting to note that clinical lesions of leprosy have not been described beyond the trachea. Muir has published three cases of leprosy of the lung but such a condition has never been seen post mortem and we believe it is of the nature of a leprosy tracheitis or possibly bronchitis and is a reaction phenomenon and only seen during lepra reaction and that all signs subside after the reaction has come under control. The mouth pharynx and larynx are involved in direct proportion to the severity of the disease. In advanced lepromatous leprosy, although clinical signs may not always be prominent bacilli can usually be found on careful examination in any of these situations. The first lesions are usually seen on the lips which may be involved in the general infiltration of the face or if the disease has extensively involved the lips they may present a large misshapen swollen appearance. On the other hand small flat topped nodules may be seen which may extend and become more pronounced. Along with the involvement of the lips the process extends to the tongue which may show no clinical signs but which sooner or later manifests gross lesions. These lesions may vary from a mild glossitis to a well marked geographical tongue with deep fissures.



Fig 101



Fig 102



Fig 103



Fig 104

Lesions of the tongue showing lesions gradually progressing to developing a mass appearance

which at times give a mosaic appearance. On the other hand actual nodulation of the tongue may be seen. The nodules are usually seen on the anterior third or tip of the tongue but may extend over the whole organ. The fissuring of the tongue which in the late stage is a prominent feature frequently extends to the lips and a painful raw cracked tongue and lips occur giving rise to much discomfort and sometimes intense pain. It should be noted that marked ariboflavinosis may be associated with advanced leprosy and therefore the amount of involvement due to leprosy or to a deficiency of riboflavin cannot be gauged except therapeutically and therefore under treatment it will be stated that it is always worth while to treat as for a riboflavin deficiency first for such treatment may ameliorate some of the worst symptoms.

The soft palate, hard palate and uvula may be all affected. The leprosy process spreads from the mouth and nasopharynx to the uvula and this may become grossly distorted bound down to the roof of the mouth or be destroyed altogether and in such instances there may be experienced definite difficulty in swallowing liquids or semi solid foods. Sometimes when the uvula appears to have been destroyed it has actually been pulled back by the fibrous tissue which always forms in late stages of nasopharyngeal leprosy and may be found pointing up towards the posterior nares. It should be remembered that ulceration of the hard palate leading to perforation occurs in leprosy and is not necessarily syphilitic in origin. Syphilis may also be mimicked by the greyish slough on the tongue which resembles leukoplakia. These conditions in leprosy therefore should be borne in mind and an anti syphilitic course should not be given unless the physician is convinced that the lesions are complicated by the presence of the spirochaete for to treat for concomitant syphilis when other signs and history of infection are absent may do more harm than good. As pointed out the Wassermann reaction does not help for this is frequently positive in advanced lepromatous leprosy.

4 Lesions of the Larynx

Leprosy of the larynx nearly always starts in the free extremity of the epiglottis. This is inflamed and small granulations are seen on its surface. From the epiglottis the disease passes to the cushion of the epiglottis the mucous membrane of the cuneiform and corniculate cartilages is involved and the disease extends to the false cords. While nodules on the true cords are sometimes seen these are usually involved secondarily as a result of their becoming embedded in the surrounding granulated tissue.

It is frequently extremely difficult to see the larynx in leprosy by ordinary indirect examinations for the epiglottis may be so enlarged inflamed or fibrotic that a view of the larynx is quite impossible. The best technique for laryngeal examination in leprosy is to have the patient sitting in front of the physician on a slightly higher stool. The patient opens his mouth wide bends his head slightly forward. The tongue is grasped with a piece of gauze and gently pulled forward. The mirror is inserted well back and the tongue slightly depressed. This manoeuvre sometimes overcomes the difficulty of an enlarged epiglottis. Those who are proficient and dextrous will find if the larynx cannot be seen because of an unduly large epiglottis that after cocaineising the throat the epiglottis can be tilted out of the way by a retractor and a better view of the larynx obtained. As the importance of recognising early lesions of the larynx is so great physicians taking up leprosy as a speciality would be well advised to become proficient in the newer methods of direct laryngoscopy for leprosy of the throat has been too often passed over as hopeless and no real attempt has been made as yet

except in Carville Louisiana really to study such conditions and attempt to ameliorate the symptoms or retard the progress of the disease

Leprosy of the larynx is seen in two forms

- 1 Fibrotic
- Ulcerative

In the fibrotic form the tissue of the larynx reacts to the presence of the M leprae by a fibrous tissue reaction which produces immobility of the cords and so hoarseness of the voice The disease is slowly progressive and may lead to complete stenosis of the larynx A patient may carry on for years with little or no discomfort and die of some complicating disease before distressing symptoms of laryngeal stenosis occur

The ulcerative form is of very much more serious import For in this condition granulatous tissue develops rapidly and there is a considerable increase of secretion giving rise to hoarseness pain and breathlessness Punched out ulcers form over the mucous membrane of the arytenoid and corniculate cartilages and in the region of the false cords and these become covered with a greyish or yellowish slough The danger to life in such cases is considerable for in the first place due to the extensive inflammatory involvement oedema of the glottis may suddenly arise and the patient die in acute distress or as a result of the rapidity of spread of the granulatous process the rima glottidis may become so narrowed that signs of suffocation and distress arise necessitating an immediate tracheotomy Enough has been said to emphasise the need of constant vigilance in lepromatous leprosy The nose throat and larynx should be examined frequently and active palliative measures undertaken on the first appearance of any clinical signs of involvement of these structures Leprosy of the nose mouth pharynx or larynx is of such serious import that delay may result in much misery and distress to the patient It is these manifestations of advanced leprosy in the nose and throat that make the disease so feared and shed a pall of terror over it but the physician must endeavour to retain a sense of proportion and bear in mind that it is only a small proportion of all cases which manifest these advanced and almost terrifying aspects of the disease

LESIONS OF THE EYE

The eye is one of the most valuable organs man possesses and is liable to be attacked in leprosy Therefore every patient suffering from leprosy should have a routine examination of the eye and this should be repeated at frequent intervals especially if there is any doubt as to the condition of any of the elements of the eye If active disease is discovered vigorous measures must be instituted at once Too often a defeatist attitude is adopted It must not be forgotten that careful observation intelligent and active treatment will prevent blindness in many cases and in others stave off that tragedy for a number of years The eye can be affected both in neural as well as lepromatous leprosy

1 Involvement of the Eye in Neural Leprosy

In neural leprosy the eye becomes secondarily affected owing to the involvement of the nerves supplying the muscles of the eye and cornea Lesions are produced as a result of the ensuing paralysis due to the invasion by M leprae particularly of the Vth nerve and the ophthalmic division of the Vth nerve When the Vth facial nerve

■ involved paralysis of the orbicularis muscles ensues and lagophthalmus results. When the first division of the Vth nerve is similarly involved anaesthesia of the cornea is a prominent sign. Because of the anaesthetic cornea irritant particles get into the eye without being noticed. If there is associated lagophthalmus the blinking reflex is lost and the lower pole of the cornea is uncovered when the patient is asleep. In consequence inflammation sets in due to dust and wind irritation which shows itself in conjunctivitis and later the inflammatory reaction may so damage the cornea that actual ulceration follows. Further owing to the lagophthalmos which is liable to occur the eye cannot be closed and when the patient attempts to close his eye in sleep the cornea remains uncovered and the delicate tissue becomes further devitalised. There then appears an inflamed area at the lower pole of the cornea which at first simulates an arcus senilis but goes on to a soft mushy inflammatory mass with enlarged vessels. This becomes organised and a dense opacity is the end result causing gross impairment of sight and the infective process may extend (? pyogenic) to the iris. Thus the iris becomes secondarily involved is bound down to the scar and all the signs of chronic irritative iritis or iridocyclitis ensue. Hence the eye lesions in neural leprosy are solely due to mechanical causes following paralysis of muscles and anaesthesia of the cornea and are in no way specific.

■ Involvement of the Eye in Lepromatous Leprosy

The disease may extend to the eye in two ways

(a) By a process of spread probably through the lymphatics or directly through the naso lachrymal duct or over the lid margin to the conjunctiva.

(b) Through the blood stream. The ciliary body appears to bear the brunt of the infection.

In one or other of these ways leprosy may affect the eye and all elements become invaded. The different structures of the eye that become affected will now be described.

(i) SCLERA

Leprosy appears to affect the sclera in two ways

- (a) A diffuse lepromatous involvement
- (b) A circumscribed lepromatous involvement



FIG 10 — Diffuse ep scleritis

(a) Diffuse Involvement

The sclera may be involved without clinical signs for in a certain proportion of cases smears taken from the sclera in lepromatous leprosy may show M leprae. Diffuse involvement of the sclera in lepromatous leprosy is seen in two forms

- (i) Acute
- (ii) Chronic

(i) Acute. The acute form usually is associated with signs of the involvement of other elements of the eye e.g. iris. The whole sclera and conjunctiva are affected and

there is much inflammation and pain. It is inconceivable that a process of this nature should confine itself to the scleral tissues alone and it is almost certainly associated with extension of the disease from the ciliary body to the choroidal coat of the eye.

(1) *Chronic* This is a very common early sign of eye infection. The eye looks quiet and there is no evidence of acute disease: the cornea is clear and the pupils regular and react well, but on examination especially in the region of corneo scleral junction more often laterally than medially is seen a discolouration of the sclera and signs of mild inflammation. This involves the episcleral tissue and the spread is probably from the conjunctiva. The affected portion of the sclera is of an orange colour and there is an increased vascularity. Scrapings from the area will show numerous acid fast bacilli. This condition may go on for months and years and never extend and clear up if the disease becomes quiescent. On the other hand the process may become acute and not only cause pain and marked inflammation but extend to the iris and ciliary body resulting in an iridocyclitis. Still another possibility is that the affection may extend throughout the sclera and a circum corneal inflammation is noticed. The lepromatous tissue causing a certain amount of oedema gradually invades the cornea and obliterates in time the whole cornea. When the process is as active as this none of the elements of the eye escape and blindness ensues. It is in these cases one may see owing to thinning of the sclera tissue actual staphyloma.



Fig. 106—Nodule on the sclera removed by operation

(b) 1 *Circumscribed Lepromatous Involvement*

The infection here confines itself to one area and a lepromatous nodule develops. This shows itself in a firm fibrous nodule which can be removed by operation. On the other hand the nodule may be soft and more vascular and difficult to remove because it is impossible to define the borders of the leproma accurately.

(2) CORNEA

The cornea is very commonly affected with leprosy and the disease is seen in four forms:

- (a) A superficial keratitis
- (b) Pannus formation
- (c) Deep keratitis
- (d) Corneal ulceration

(a) 1 *Superficial Keratitis*

This may commence as small white dots on the cornea or grey spots which coalesce and extend gradually involving the whole cornea. No pain may be felt but when the process begins to spread the patient may complain of a mistiness of vision. Vision is better in bright light than in dull light and spots appear in front of the eyes.

Though superficial keratitis is first seen in discrete spots the whole area occupied

by the spots is inflamed. Slit lamp examination will reveal this. In the early stages there may be no other signs of inflammation.

(b) *Pannus Formation*

In many cases the maculae are large and irregular, blood vessels cross over the limbus on to the cornea and these under the slit lamp have been described as finger like processes. Gradually a leprotic pannus is formed. At other times the small spots coalesce and form a diffuse opacity. Along with the spread of the process in the cornea further activity of the disease may be seen by the appearance of an exudate into the anterior chamber and extension of the inflammation to the iris and ciliary body. It must be remembered that superficial keratitis is usually very slowly progressive and with active measures the progress of the disease is often arrested, sometimes permanently at other times for several months or possibly years.

(c) *Deep Keratitis*

This is a serious form of the disease and is a deep seated inflammation probably spreading from the ciliary body and is difficult to arrest and frequently extends over the whole cornea causing complete blindness. Much can be done for the condition but continuous watchfulness must be maintained for there is no pain and the patient if ignorant does not realise the progressive nature of the trouble and inevitable blindness unless treatment and great care is exercised. It must be pointed out that when the cornea is affected this may be the only outward manifestation of leprosy of the eye but nevertheless all structures of the eye are involved and bacilli have by this time spread by lymph and blood stream to every part of the organ.

(d) *Corneal Ulceration*

This is liable to occur when there is extensive involvement of the eye especially in the more acute conditions and necessitates careful observation and active and immediate treatment. Unless treated vigorously a fibro purulent exudate is liable to occur in the anterior chamber but there is seldom free pus. The scar resulting from such lesions gives rise to a dense opacity and great impairment of vision.

(iii) IRIS

Leprous infection of the cornea and sclera may be the result of blood stream infection of the ciliary body or the consequences of direct spread from surrounding tissues or through the nasolachrymal duct. Iritis which occurs comparatively frequently is probably a blood stream infection. This is almost certainly the case in acute iritis. Leprosy can involve the iris in three ways

- (a) Acute iritis
- (b) Subacute iritis
- (c) Chronic iritis

(a) *Acute Iritis*

This may supervene on a subacute iritis but frequently the first indication that the eye is affected in leprosy is a sudden attack of acute iritis. The usual symptoms are present viz photophobia, pain and inflammation. On examination the iris loses its lustre, the pupil is fixed and small and the light reflex lost. The iris becomes muddy

there is intense circumcorneal inflammation and there may be oedema of the conjunctiva. If vigorous treatment is not instituted permanent damage will result. In the acute stage there is danger of a fibro purulent exudate forming in the anterior chamber but as stated above free pus is seldom seen. At the end of the attack if adequate treatment has not been given permanent damage to the iris follows.

(b) Subacute Iritis

This usually follows on several attacks of acute iritis or from the start the infection may be a smouldering one with dull pain slight photophobia and fixed pupils. A characteristic of subacute iritis in leprosy is said to be white spots on the iris. These are seen frequently but are not constant. The iris is irregular and bound down by the inflammatory process and unless active and vigorous treatment is instituted for the condition it will pass on to chronic iritis.

(c) Chronic Iritis

This may be quiescent or mildly active so long as there is photophobia lachrymation or pain the inflammation is active. In the chronic stage the pupil is completely bound down by adhesions and a dense pupillary membrane is formed which is impossible to break down by therapeutic means. The extent of loss of vision depends on the amount of occlusion of the pupil.

It is an extraordinary phenomenon that in spite of the complete occlusion of the pupil at times glaucoma seems to be a rare complication in a chronic iritis of leprosy. In acute iritis however an acute secondary glaucoma may be seen due to blocking with the inflammatory products of acute iritis but in the Lady Willington Leprosy Sanatorium during a period of ten years only half a dozen cases of glaucoma have been seen although there is a large number of patients with dense pupillary membranes and partially or completely occluded pupils due to past attacks of iritis. This is probably due to the fact that the brunt of the attack in leprosy of the eye is borne by the ciliary body which in all probability is destroyed.

Any structure of the eye may become involved and iritis never occurs without corresponding affection of the ciliary body resulting in what is really an irido cyclitis. No fundus changes have been described in leprosy but that may be because by the time the fundus is involved the cornea is so affected or the iris so bound down that it is impossible to see it. In cases where the disease affects the iris it becomes very brittle and an iridectomy may be an extremely difficult operation.

Opacity of the lens resulting in cataract is comparatively common and the chronic inflammatory condition of the structures around the lens must be a predisposing factor in the formation of this condition.

As has been pointed out when lepromatous leprosy extensively affects the eye all the elements of the eye become involved and the lepromatous process may then invade



FIG 107 - (c) Iritis in leprosy
white spot on iris
= leuk.

the anterior chamber resulting in the formation of exudates. Along with the iritis and irido cyclitis patients may get periodic attacks of eye pain which are sometimes of great severity. The attacks pass off and may recur at varying intervals and indicate extensive involvement of the whole uveal tract. Unlike pan ophthalmitis due to sepsis there is no oedema of the eyelids.

For those who wish to study in detail the changes in the iris in leprosy the excellent monograph by De Barros (1936) and reprinted in the *International Journal of Leprosy* (1940) should be consulted.

Leprosy of the eye is a rich field for investigation especially in connection with the study of the various types of iritis and of the fundus in cases of generalised leprosy with clear ocular media. Further ophthalmologists who wish to study the pathological conditions which affect the eye could not do better than spend 3-6 months in an intensive investigation of eye lesions in a modern leprosarium.

CHAPTER VII

TREATMENT OF LEPROSY

Introduction

Probably there is no disease which calls for more ingenuity in the matter of treatment on the part of the physician than leprosy. So long as there is no specific treatment for the disease the therapeutics of leprosy will remain one of considerable difficulty. Nevertheless while in many ways the disease is hard to treat and the results very variable and not a little disappointing yet a proper understanding of the therapeutics of leprosy will result in the relief of a large number of sufferers and the amelioration or prevention of the more distressing symptoms of the majority. The physician treating a case of leprosy must maintain a reasonably hopeful outlook even when the chances of complete recovery are limited for constant vigilance, careful treatment and a cheerful outlook will gain the confidence of the patient and do more than anything else to help recovery. In a disease whose treatment is so little understood the psychological outlook both on the part of the patient and of the physician is of extreme importance. The influence the mind has over the body in disease is receiving ever increasing attention and in leprosy where the mental reactions are so great the importance of a well balanced mental approach cannot be too strongly stressed. It is just here that the challenge particularly comes to the Christian doctor for he is equipped in a special way to deal with the many difficulties patients have to face when they are afflicted with a chronic and largely incurable disease. I feel I must at this point state quite emphatically that without a firm belief in Christ I should find it difficult to give any kind of answer to the problem of suffering and without some answer to this age long riddle it is impossible to approach the treatment of a chronic and to some extent greatly mutilating disease in a frame of mind which will enable the patient successfully to face the long uphill road to renewal of health.

In considering the therapeutics of leprosy a clear understanding of its pathology is necessary and it is to be remembered that in those forms of leprosy where there is active defence the body is dealing quite effectively with the disease and the physician's task is to minimise any damage which is liable to follow as a result of this defensive process. Therefore in treating leprosy it is not only a question of treating the disease but of preventing if possible its further progress and at all times the physician has to ask himself What special measures must be taken to overcome any deformity which is likely to ensue? Hence it will be readily understood that the therapeutics of leprosy is a subject which is not only complicated but requires long experience and patience if success is to be attained.

Much has been said about the necessity of dealing with predisposing diseases. While it is only reasonable to treat any concomitant infection or complicating condition too much reliance should not be placed on the results after this has been done for as pointed out in the earlier chapters the progress or retardation of leprosy seems to bear little relationship to the general health of the patients and in the past predisposing diseases and debility of the patient have been put forward as the causes for lack of success of treatment when all along the real reason has been our ignorance of more efficient and

active therapeutic measures. Nevertheless because leprosy is a chronic disease of long duration patients frequently suffer from concomitant disease both acute and chronic besides they have the ordinary liability to parasitic infections of all kinds. Therefore any complicating condition should be treated and as far as possible eliminated. Generally speaking if the complicating condition is acute special treatment should be withheld but in the more chronic diseases (e.g. syphilis) these may be treated along with leprosy. In the case of tuberculosis and leprosy because the former is a comparatively more serious and acute disease one's whole attention should be concentrated on it rather than on the leprosy.

With regard to special remedies it cannot be too strongly stressed that no departure should as yet be made from the well tried and accepted derivatives of hydnocarpus oil. The general public and not infrequently the medical profession are often deceived by extravagant claims of firms who wish to bring to the attention of the public special drugs of their own. Two principles should be remembered—avoid the newest remedies and avoid the more expensive remedies.

Before the general principles of administration of hydnocarpus oil are described it might be of interest and of some value to refer to remedies which have from time to time been in vogue in order to illustrate how many remedies have been used and emphasise the care needed before any therapeutic claim can be established. At the end of the nineteenth century Radcliffe Crocker advocated mercurial preparations. This is of interest because the ingredients of many of the Indian indigenous remedies contain mercury and this possibly accounts for the frequency of reactions after treatment by persons practising indigenous medicine. A few of the more important of the remedies which have been or still are in vogue will now be discussed.

Potassium Iodide

Our general experience with this remedy is such that its use is not advocated. In skilled hands and in carefully selected cases there may be some benefit but in a disease in which there are so many variable factors and which ordinarily shows periods of remission it is almost impossible to say whether in the few cases which show favourable results that some other factor is the cause for the improvement and not the drug. It has been said by the advocates of potassium iodide that treatment can be controlled by the sedimentation index test but as has been pointed out this test in our opinion is not a sufficiently reliable one on which to depend in the control of treatment. Potassium iodide is not recommended because of the danger of its precipitating reactions even in small doses. Therefore it is better avoided altogether. No prescription in the Lady Willingdon Leprosy Sanatorium contains potassium iodide. Anyone who has had considerable experience in an institution will bear out the danger of its administration for not a few patients have been crippled as a result of reactions in nerves and blinded through the flaring up of eye lesions. It is therefore advisable to avoid iodides entirely in the treatment of leprosy or of any concomitant diseases which are ordinarily treated with iodides e.g. bronchitis, syphilis etc.

Some advocate provocative doses of iodides in order to produce a nasal discharge in the hope that a diagnosis may be made. This in our opinion is entirely unjustified. Firstly because nasal lesions are seldom or never primary, secondly because of the danger of precipitating reactions.

It should be remembered that potassium iodide has from time to time been used

in leprosy and that the father of modern leprosy, Armour Hansen, recognised its dangers and stated in his classical monograph that 'even small doses of iodides produce new eruptions and patches'.

Arsenic

Preparations of arsenic were used by the earlier physicians and the late Dr Danielssen used it in the form of Fowler's solution in increasing doses and came to the conclusion that the apparent good was due to the emaciation produced but when that was recovered from the nodules gained their previous size! Arsenic was re-introduced by Hasson (1922) but has again rightly fallen into disfavour.

Antimony

Antimony preparations have been extensively used especially by Gawston (1920). Rodriguez and Kubinus (1923) carried out a series of experiments on the value of antimony in leprosy and came to the conclusion that antimony appears to be of no therapeutic value as a means of treating leprosy. In our experience as will be pointed out later antimony is only of value in lepra reaction and should not be used except for this condition.

Copper

Within recent years copper in colloidal form has been extensively used but these preparations are now in dis-favour for there is no conclusive evidence that they are of any general value in the treatment of leprosy. In so far as it appears that heavy metals in small doses tends to control reactions the value of copper may lie in this direction but there are other and safer remedies which can be used and therefore copper and its derivatives are not recommended.

Sera and Vaccines

As far back as 1890 Carrasquilla recommended serum therapy. Since then there have been many advocates of serum or vaccine treatment and the best known are Rost (1910), Deycke, Williams and more recently Reinsterne (1936) have all advocated preparations of vaccine and sera but as no serum or vaccine has been of proved usefulness there seems little value in detailing the various acid fast organisms used in their preparation or in describing the treatment in detail. Sufficient is it to say that all such remedies have largely gone out of favour and as far as it is known are not used now in India or the East.

Aniline Dyes (Trypan Blue, Brilliant Green, etc.)

Some years ago aniline dyes were advocated for the treatment of leprosy. In this connection it is interesting to note that these substances are employed in the technique of intra vital staining of the macrophages or wander cells of the reticulo-endothelial system. Many in this group having strong antiseptic properties it was therefore reasonable to conclude that they would have an effect on the *M. leprae*. In the fair skin it was interesting to note that the lepromatous lesions of leprosy were stained the colour of the dye used showing that the substance was absorbed by the lepra cells in the corium. However although some cases showed improvement there was no evidence that the dyes had any effect on the bacilli themselves. Thus once again emphasises the parasitic nature of *M. leprae* and hence the difficulty of attacking it by ordinary antiseptics.

The aniline dyes like so many remedies are now no longer recommended in the routine treatment of leprosy

Sulphonamides

As the sulphonamide products were certain to arouse hopes in the treatment of leprosy a trial of sulphapyridine—acknowledgments are due to Messrs May & Baker for generous samples—was undertaken at the Lady Willingdon Leprosy Sanatorium in 1940 and after six months the experiment was abandoned and the following is a summary of the experiment. Nine cases in all have been treated. Two patients had two courses of treatment one patient had three courses and the other six had one



FIG 108—Acute reaction after administration of M & B 693 in lepromatous leprosy



FIG 109—Reaction subsided after stoppage of drug and patient improved on standard treatment and was subsequently discharged as negative

course. Early in the experiment it was found that the administration of M & B 693 tended to precipitate lepra reaction with in some cases high fever. In one case this was accompanied by actual ulceration of the lesions. The initial dose was two tablets twice a day increasing to six tablets but it was found that few patients could tolerate a dose of six tablets continuously and that nausea and vomiting ensued. The average dose was therefore reduced to three tablets in some instances and four in others. This dose was maintained until a reaction occurred. When the fever was definitely established the drug was stopped and the antimony products administered to control the fever. In those who had more than one course a month's rest was given and then M & B 693 was resumed in the same dosage. In addition to this sulphonamide in the form of a paste was used for certain types of ulceration. The following conclusions were arrived at

(a) The remedy is liable to produce acute lepra reaction and in some cases this is exceptionally severe

(b) There is no evidence that the patient's leprosy condition is benefited by M & B 693

(c) There seems to be a greater tendency for nausea, alterations in pulse rate and drug dermatitis to occur in leprosy patients than in persons suffering from other diseases

(d) The sulphonamide preparations in the form of a paste have a decided place in the treatment of certain types of trophic ulcers

(e) It has been noted however that where M & B 693 or other sulphonamide is specifically indicated (e.g. pneumonia or septic conditions) it can be given without difficulty

(f) The sulphonamides appear to have no influence on leprosy and should not be used unless there are specific indications for its use

Since this work Dharmendra (1944) and his colleagues have carried out experimental work on sulphapyridine in rat leprosy with the following conclusions

Under the conditions of the experiment described sulphapyridine has failed to modify the course of experimental rat leprosy in white rats. The drug does not appear to have an inhibitory effect *in vivo* on the *Mycobacterium leprae muris* although *in vitro* it has been known to have a bactericidal effect in a 1 in 1 000 dilution

Diphtheria—Formal Toxoid

Oberdoeffler and Collier (1939) following the development of the theory that a tuber of the *Colocasia antiquorum* family eaten as a staple diet was an epidemiological factor in the requirement of lepromatous leprosy suggested that the administration of diphtheria anti-toxin or of diphtheria toxoid might be an effective treatment of leprosy. Cochrane (1941) and Chatterjee (1942) have not confirmed this work and Wade (1941) and Low (1942) both appealed for further trials. As far as is known the administration of diphtheria toxoid has fallen into disuse and is not now recommended. The earlier hopes of these workers have been shattered by more recent work.

Promin and Diasone

The latest remedies to be advocated are the new products related to the sulphonamides—the promin group. Iaget (1944) reported recently that promin in massive doses was more effective than any other treatment at present known for leprosy. Readers are warned however not to use such remedies until they have been much more extensively tried out under adequate experimental conditions. The author has recently had the privilege of personal conversation with Iaget and his co-workers and while he maintains a cautious attitude considers that the results are significant and that these remedies deserve a further and more extensive trial. Iaget and his co-workers state that promin or its one or promizole is equally effective in treatment.

A word may not be out of place on the question of the technique of testing remedies claimed to be effective in leprosy. In the first place before any physician can evaluate a given drug he must be able to diagnose the type of disease accurately and confirm his diagnosis histologically. The more active tuberculoid cases and especially the large group of atypical intermediate or border-line cases sometimes simulate lepromatous so closely that confirmation of the classification by biopsy is essential. The wisdom of this will be understood if it is recalled that two or three atypical cases in a small group chosen for a therapeutic experiment may vitiate the whole test. Further an apparent

dramatic recovery becomes so firmly fixed in the mind of the physician that he is apt to draw conclusions from the exceptional rather than the general. Hence one should not undertake the testing of new remedies seriously unless firstly there are facilities for histological work and a person sufficiently experienced to interpret the sections and secondly the remedy should be under trial from six months to two years. Six months will be sufficient to disprove a remedy but confirmation of initial favourable results needs at least two years. If every one would avoid publishing the results of treatment for two years much disappointment would be avoided for it is to be remembered that the person with advanced lepromatous leprosy is only too ready to grasp at any therapeutic straw and it is both unkind to the patient and unscientific to publish the result of remedies either not properly tried or tried for too short a period on only a few patients. In any case when in doubt only lepromatous cases established histologically should be used to test out new remedies.

Diet in the Treatment of Leprosy

The question of diet is often raised. All that can be said at present is that a general all round well balanced diet should be given. In children skimmed milk is of value if there is a vitamin B deficiency and in adults who complain of bone and nerve pain wheat in place of rice is of definite benefit. Eight ounces of skimmed milk (Acorn brand) i.e. one ounce of powdered milk to eight ounces of water should be prescribed whenever there is evidence of angular stomatitis. As has been stated there is no conclusive evidence that special diet affects any condition other than the deficiency for which it is given. There is little proof that dietary improvements make any substantial difference in the treatment of leprosy.

Chaulmoogra Oil in the Treatment of Leprosy

As has been stated the only remedies which have been consistently advocated over the past two decades have been derivatives of chaulmoogra oil and while these are not claimed to be specific they have not as yet been bettered and it is recommended that the hydnocarpus (chaulmoogra) derivatives be used as the treatment of choice. Their method of action is still unknown. The following theories have been suggested.

- 1 Hydnocarpus preparations dissolve the fatty capsule and liberate bacilli so that they can be dealt with in other words as Mur used to say of such drugs they may act by lepromalysis.

- 2 There may be still an undiscovered active principle which exists in such small quantities so that large doses must be administered for it to have any effect.

■ Intradermally it may act in some way on the nutrition of the bacilli and render the environment unfavourable to its multiplication.

A study of the chapters on Pathology and Immunity will indicate to the reader that an investigation into this latter reason may be a most profitable line of future research. We believe that if the nutritional state of the tissue comprising the corium could be so altered that *M. leprae* could not multiply then leprosy would cease to be a progressive disease for as already stated I am of opinion that the *M. leprae* cannot multiply and cause active disease unless they are disseminated via the skin. It is suggested that research might be concentrated on the skin and on methods to affect the nutritional state of the bacilli in the corium. This method of research falls into line

with the modern theories of the sulphonamide group of drugs because it is generally held that it is the environment which is so altered that the bacilli can no longer multiply and not a specific bactericidal action which makes these drugs so effective to certain bacteria.

The only conclusion at present drawn is that no substance as effective as the hydnocarpus derivatives has as yet been definitely discovered and therefore unsatisfactory as many of the results are these are the only remedies which can at present definitely be recommended for the routine treatment of leprosy.

Hydnocarpus Oil (Chaulmoogra Oil)

The oil from the following species has been used in the treatment of leprosy

Hydnocarpus wightiana (West coast of India)

Hydnocarpus anthelmintica (Siam and Burma)

Seraptogenus kurzu (*Hydnocarpus kurzu*) (Burma)

Cynocardia odorata (false chaulmoogra) (Burma)

Of the above remedies the only preparations which are universally used are *Hydnocarpus wightiana* and *Hydnocarpus anthelmintica*.

Hydnocarpus oil must be extracted from fresh seeds and by the cold process; good oil should be of a golden colour with no sediment and have an optical rotation of 90 degrees and an acid content of not more than 0.1 per cent. Usually clear golden coloured oil is not irritant. Milder degrees of irritation can be overcome by treating the oil with activated charcoal. The final test of good oil is whether it gives pain on injection. The following preparations of the oil are in use:

- (i) Pure *hydnocarpus* oil
- (ii) Esthers
- (iii) Salts of the fatty acids of *hydnocarpus* oil the one most commonly used is sodium *hydnocarpate* (Alepol—Burroughs Wellcome)

The method of administration of the *Hydnocarpus* preparation has varied and the following routes have been advocated from time to time

- 1 Oral
- 2 Subcutaneous
- 3 Intradermal
- 4 Intramuscular
- 5 Intravenous

The generally accepted methods are by subcutaneous, intramuscular or intradermal injection. *Hydnocarpus* oil by mouth appears to have a limited application. Intramuscular injections are inconvenient and intravenous medication should never



FIG. 110. Method of giving intramuscular injection in leprosy. Note the small wheal raised at the point of injection.

be employed as a routine method and certainly never where oily solutions are in use

For ordinary routine treatment two preparations are to be recommended

- (i) Hydnocarpus oil with 0.5 per cent creosote
- (ii) Ethyl esters of hydnocarpus oil in the following proportions

Ethyl esters	75 parts
Olive oil (or hydnocarpus oil)	25 parts
Creosote	1 part

Iodised esters and iodised oil have been advocated from time to time. Great care is necessary in the preparation of these remedies and readers who wish to use them are advised to procure them from reputable firms for facilities in the ordinary laboratory of an average leprosy institution are usually not sufficiently developed to undertake their preparation. On the other hand the preparation of ethyl esters is a comparatively simple matter and details of the preparation are added as an appendix to this chapter. It may be helpful to those who are not in touch with reliable sources of supply to indicate firms whose products have been proved to be reliable. Generally speaking it is advised to order the pure hydnocarpus oil direct from the west coast of India and Messrs Ernakulam Trading Co. Ernakulam supply a pure oil. The oil is pure if golden yellow colour without sediment and perfectly clear. Oil which is in any way opaque or cloudy with a sediment should be rejected. It is preferable to transfer all oil into glass containers and not retain it in the tins in which it is dispatched. This procedure prevents the formation of irritant products due to oxidation.

Ethyl esters of good quality iodised or creosoted (undistilled) can be had from Smith Stanistreet Calcutta or De & Co. Calcutta or from the Madras Medical Stores. The former two firms prepare a satisfactory iodised ester.

Before the war (1939-40) the laboratories of the International Institute of Leprology Rio de Janeiro prepared an excellent product of iodised esters which in a limited trial at the Lady Willingdon Leprosy Sanatorium (1940) gave promising results. A word of caution must be issued with regard to the administration of iodised products. These must be injected at once and the balance never put back into the original bottle and on no account must moisture be allowed into the esters. Exposure to air for any length of time and the introduction of minute particles of water result in an intensely irritating product. While this remark applies particularly to the iodised esters of hydnocarpus oil they are generally applicable to all preparations in which there is iodine in loose combination. To a lesser extent the creosoted oil and esters are sensitive to air and moisture and on no account should any surplus be returned to the original bottle.

In addition to the above firms Messrs Burroughs Wellcome London supply creosoted hydnocarpus oil creosoted and iodised esters under the trade name Moogrol and 'Iodised Moogrol'.

Hydnocarpus oil should be sterilised and given subcutaneously or intramuscularly and the ethyl ester mixture given intradermally. The subcutaneous route is preferable to the intramuscular but not more than 3 c.c. should be injected in one place. The higher dosages should be divided the places of choice are the outer aspects of the thighs arms and buttocks. The following is the technique for intradermal injections as advised by Muir (1930).

'An all glass or record syringe with a fine needle fitted with a guard is used. The

skin having previously been painted with spirit or iodine the needle is then introduced into (and not under) the skin. The thickness of the skin varies in different parts of the body where it is thin the needle is introduced in a sloping manner so as to form an acute angle with the skin. Where it is thicker then it is preferable to insert the needle at right angles. With a little practice the technique is soon mastered and one finds little difficulty in injecting into the corium and not into the subcutaneous tissues. An amount of the drug is injected so as to raise a weal of about one third of an inch in diameter. If a large area is to be injected it is completely infiltrated: some thirty punctures are necessary to inject about 3 c c of the esters. Each injection should be separated by about a quarter of an inch: about six to twelve punctures are needed to inject 1 c c.

No mention has been made of the sodium salts of hydnocarpus oil. The best known goes under the trade name of Alepol (Burroughs Wellcome). It is our belief that these remedies are not effective in the treatment of leprosy. They may cause irritation subcutaneously and ultimately result in blockage of the veins if given intravenously.

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TREATMENT OF LEPROSY—*continued*

WHILE it was pointed out in the previous chapter that little is understood concerning the part concomitant diseases and debilitating conditions play in the therapeutics of leprosy, it is wise to deal with such conditions, adequately treat them and where possible eliminate them. In any discussion on treatment the subject naturally falls under the following heads:

- 1 Treatment of neural cases
- 2 Treatment of lepromatous cases
- 3 Treatment of children

I Treatment of Neural Leprosy

It is a well known fact that neural leprosy particularly, has a great tendency to spontaneous cure or disappearance and that only a small proportion of such cases are liable to become lepromatous. Further to understand the treatment of leprosy especially neural leprosy, we must bear in mind what was said with regard to the pathology of leprosy in respect to tissue reaction that every reaction of the tissues to invasion by *M. leprae* is defensive. In certain forms of neural leprosy this reaction is seen in successful defence and therefore if the body is dealing with the organism satisfactorily there may be no need for us to interfere with excessive treatment and in many cases no treatment at all may be the best advice that can be given to a patient. However until the practitioner in the tropics is really familiar with the vagaries of the disease a wise principle is when in doubt—treat.

The treatment of leprosy will now be discussed for each separate type and subtype

Simple Macular Lesions

The method of treatment is both local and general. The hypopigmented areas should be stimulated as much as possible in order that pigment may be encouraged to form. There are two methods of doing this:

- 1 Painting the lesions with a counter irritant
- 2 Intradermal injections

1 *Trichloroacetic Acid* Patches should be painted with this acid but it must be remembered that it is an irritating acid and unless care is exercised ulceration and actual keloid formation may result. Therefore only a small area should be painted at a time and the acid should be allowed to dry before more is again put on. When dry the appearance of a fine dusting powder should be seen. If the appearance is that of ash then too much has been put on and the acid should be neutralised with moist sodium bicarbonate. Only a few patches should be painted at one time.

2 *Intradermal Injections* If the patches are not responding to trichloroacetic acid or if the progress is not sufficiently encouraging then intradermal injections should be given into the lesions as well. Between painting the patches with trichloroacetic

acid there should be an interval of ten days and no lesion should be re-intradermally under one month and preferably six weeks. While one set of lesions are painted with trichloroacetic acid another set is given intradermal injections. Intradermal injections tend to stain the skin but this discolouration though slow in disappearing will fade in six months to two years. Discolouration of the skin however is not a counter-indication to intradermal injections. The great tendency for hyperpigmented spots to appear after intradermal injection should be borne in mind when injecting the face and especially is this the case in the treatment of women. Because there is little evidence of the benefit neural cases receive apart from measures which involve counter-irritation it is considered advisable to commence treatment by giving intradermal injections first into all the lesions, commencing with those showing most anaesthesia. After the patient has become used to the injections then the other lesions can be injected. The commencing dose for intradermal injections of the ethyl ester mixture is 1 c.c. increasing by 1 c.c. each week until 5 c.c. are injected. Many workers advocate subcutaneous injection of hydnocarpus oil and these may be given in addition to the intradermal injections of the esters. It is recommended however that the patient be first given intradermal injections and when he has reached a dose of 5 c.c. intradermally subcutaneous injections of hydnocarpus oil and 0.5 per cent. of double distilled creosote be given. These injections may be given on the same day or if more convenient on another day of the week. The commencing dose after 5 c.c. of ester is reached is 1 c.c. increasing by 0.5 c.c. each week until 10 c.c. is given. It must be remembered that 5 c.c. of the esters mixture is continued all the time the hydnocarpus injections are being given and therefore the maximum dose is 15 c.c. per week. When this dose is reached it is given ten times after which the patient is given a month's rest. After a month's rest intradermal injections are started at 2 c.c. increasing by 1 c.c. each week up to 5 c.c. then as before if felt desirable hydnocarpus oil injections are commenced again starting at 2 c.c. and gradually reaching the maximum dose of 10 c.c.

II Treatment of Minor Tuberculoid Leprosy

It is to be borne in mind that all minor tuberculoid leprosy is a form of active tissue response and when there is one or at the most only two lesions of small size then the best procedure is to excise the whole lesion and ask the patient to return every six months for observation. When the lesion is too large to excise or there are too many then intradermal injections should be given as described until the whole patch or patches have been injected then a month's rest prescribed and treatment recommenced on the above lines until the patch has been converted into scar tissue.

III Treatment of Major Tuberculoid Leprosy

As has been pointed out major tuberculoid leprosy represents the most active tissue defence and the more active this defence that is the more acute the lesions the less active treatment is necessary. Any major tuberculoid lesions which are markedly erythematous or scaly should not be given intradermal injections. We are of opinion that in these instances no treatment with hydnocarpus oil is necessary for the tissues are effectively dealing with the *M. leprae* and therefore there seems no point in interfering. When the patches are not markedly erythematous especially in the more chronic fibrous type of lesions which may show gross infiltration then intradermal injections

at weekly intervals appear to be the most rational treatment. These are continued until all lesions are healed and the dosage is increased up to a maximum of 5 c c or to that amount which is sufficient to infiltrate all the lesions.

Some workers advocate subcutaneous injections in addition. These may be given along the lines laid down for simple macular leprosy but the writer himself sees little point in giving subcutaneous injections.

IV Treatment of Lepromatous Leprosy

The question of building up the health of the patient and the rectifying of any deficiency or concomitant disease is stressed by many writers particularly by Muir. It seems reasonable to conclude that if there are complicating factors the clearing up



FIG 111—Major tuberculous leprosy showing acute reaction with marked scaling lesion of buttock.



FIG 112—Similar lesion on face. Lesions cleared up within three months but unfortunately the patient succumbed to primary tuberculosis.

of these will naturally help in combating leprosy. But just because a patient is not responding to treatment it need not be assumed that this failure is due to some as yet unrecognised dietetic deficiency or to a poor general state of health. It is often suggested with too much facility that these causes underlie the deterioration in leprosy when the real factor is often unknown and needs more intensive study. Our inability in many cases to attain better results is largely due to our imperfect method of treatment and our still all too inadequate state of knowledge. While making such a statement the importance of a thorough physical examination and the treatment of dietetics and other complicating factors should not be overlooked. If such conditions are discovered then there may be a much better chance of recovery when these conditions are relieved. It is sometimes the strong, healthy, well built muscular adult with no discoverable predisposing cause who is most difficult to treat.

A great deal has been written with regard to diet in leprosy but there is little evidence that this plays a vital part in its treatment. A good all round diet should be aimed at where possible rice should be substituted for wheat and a proportion of sprouted gram lentil or other wholesome cereal should be added to the diet. In children with angular stomatitis sore tongue or other signs of vitamin B complex deficiency skimmed milk forms a useful addition to the dietetic regime.

While the treatment of lepromatous leprosy is in many ways disappointing yet much can be done to relieve the patient of his distress prolong the usefulness of his life and in many instances arrest the disease for several years and sometimes permanently. Nevertheless while modern treatment is in many ways not entirely satisfactory as far as lepromatous leprosy is concerned yet by careful treatment especially in the earlier cases there is a reasonable chance of the patient becoming non infective and his disease quiescent if not permanently arrested. The relapse rate however is still disappointingly high.

The sheet anchor of our treatment in lepromatous leprosy as in neural is hydnocarpus oil and its derivatives. A great number of opinions have been expressed in respect of the optimum dose of hydnocarpus oil for lepromatous leprosy. Further much thought has been given to the question as to whether intradermal or subcutaneous injections or both should be given. In spite of several years experimentation and this is still being continued no light has been shed on the question of the optimum dose of the hydnocarpus preparations. While we consider small doses under 5 c.c. of little value there is as yet no definite evidence that large doses above 15 c.c. are of any more value. Hence we recommend for routine treatment a maximum dose of 15 c.c. Further we consider the most rational treatment is by intradermal injection and we feel that the patient should start with intradermal injections and then go on to subcutaneous injections. The following tables summarising an experiment on the optimum dose give a comparison between the various methods of injection at the Lady Willingdon Leprosy Sanatorium and lend support to the contention that intradermal and subcutaneous injections are better than either alone.

TABLE IV

Negative	Much Improved	Improved	Stationary	Worse	Total
<i>Intradermal Group</i>					
4 3.0°	13 11.4°	29 23.4°	39 34.3°	29 25.4°	114
<i>Subcutaneous and Intradermal Group</i>					
15 13.0°	26 22.6°	24 20.8°	30 26.1°	20 17.8°	115
<i>Subcutaneous Group</i>					
2 3.9°	7 13.7°	2 3.9°	20 39.2°	20 39.2°	51

TABLE XV

Final Result	Average Period of Treatment		
	Subcutaneous and Intradermal Group	Intradermal Group	Subcutaneous Group
Negative	2 years 1 month	3 years 1 month	3 years 10 months
Much improved	1 year 10 months	2 years 2 months	2 years 8 months
Improved	1 year	1 year 6 months	2 years

In out patient clinics where numbers are excessive it is a sound policy only to give 2 c.c. a week to patients until they demonstrate their willingness to persevere with treatment after which a planned course of treatment as will now be described can be chalked out.

In lepromatous cases it is wise to commence treatment cautiously and therefore the commencing dose should be 0.5 c.c. of the ethyl ester mixture intradermally. In starting intradermal injections it should be borne in mind that there is generally speaking no loss of sensation in lepromatous patches hence this method of injection is sometimes very painful. Therefore the less sensitive sites should be chosen to commence with—the outer aspects of extremities buttocks. The physician however must remember that while every sympathy must be given to the patient we must encourage him to endure the pain of the injection because the battle of leprosy is only won by perseverance and a determined mind. Hence because of the nature of the disease every effort should be made to keep the patient cheerful and optimistic maintaining his morale in such a way that the patient neither loses heart in the remedies employed nor his confidence in the physician treating the case.

In starting treatment in lepromatous cases it is recommended that intradermal injections be started once a week using the ethyl ester mixture. The commencing dose is 0.5 c.c. increasing by 0.5 c.c. until 5 c.c. is reached. When the patient has got up to 5 c.c. intradermally this dose is maintained and preferably on a second day in the week at about three days interval subcutaneous injections of hydnocarpus oil are commenced. The dose is 0.5 c.c. increased by 0.5 c.c. each week until 10 c.c. is reached. That is a patient is slowly pushed up until a maximum of 15 c.c. (10 c.c. ethyl ester mixture intradermally 10 c.c. hydnocarpus oil subcutaneously) is given on two separate days in the week. This maximum dose is maintained for ten weeks and then the patient is given a month's rest. At the end of the month provided there have been no setbacks the patient commences with 1 c.c. ethyl esters intradermally going up to 5 c.c. by 0.5 c.c. weekly increments. After 5 c.c. ethyl esters is attained then subcutaneous injections of oil are again started—the dose is 2 c.c. increased by 0.5 c.c. each week until 10 c.c. is reached. In this way successive courses of treatment are pursued until all the lesions are negative. All bacilli bearing lesions are injected until the whole body is covered. An interval of a month should be allowed before the same area is again injected intradermally.

If circumstances do not permit of the patient coming twice a week for injection then when the patient is ready for subcutaneous injections they may be given on the same day. It must be borne in mind that in this case greater care must be exercised in the gradual increasing of the dosage. The maximum dosage is still 15 c.c. i.e. 5 c.c. intradermally and 10 c.c. subcutaneously.

Some writers have described symptoms of intolerance to the hydnocarpus remedies



FIG 113

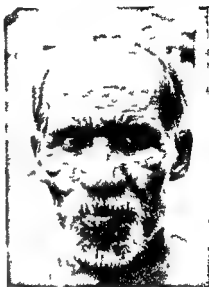


FIG 114

Advanced leprosy showing result of treatment after intradermal injection

in the form of nephritis, dimness of vision and nausea. We have had no experience of this although many thousands of cases must have been treated for prolonged periods.

V Treatment in Children

Little need be said about treatment in children except that while intradermal and subcutaneous injections should be given to children there is an age limit and most workers feel that children of seven and under are hardly likely to stand up to such methods of treatment. It is always advisable to treat children away from their parents and preferably along with other children. It is remarkable how even little children will stand up to and will ask for intradermal injection when the environment is favourable and the doctor of an understanding nature.

In children of five years and under hydnocarpus oil by mouth is worth a trial although no claims are made for this method. Children from 2-4 years should commence with 1 minim t.d.s. in a little milk or a lump of sugar and this dose should be slowly increased until the patient is given 3 minims t.d.s. In those cases where the environment is such that a child will not take injections then oil by mouth may be given and the dosage for children from five years of age to seven is 7 minims t.d.s. The maximum

TABLE XI

Final Result	Average Period of Treatment		
	Subcutaneous and Intradermal Group	Intradermal Group	Subcutaneous Group
Negative	2 years 1 month	3 years 1 month	3 years 10 months
Much improved	1 year 10 months	2 years 2 months	2 years 8 months
Improved	1 year	1 year 6 months	2 years

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FIG 113

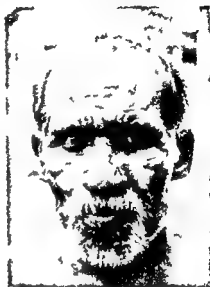


FIG 114

Adapted from J. L. Propoy, 'Long results of treatment after extensive intradermal injections'

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TREATMENT OF LEPROSY—*continued***I Control of Treatment**

One of the most difficult tasks of a physician in undertaking the management of a case of leprosy is to know when to withhold special remedies. The question of the treatment of reaction will be considered later but what are the indications pointing to the necessity for stopping or reducing the dose of hydnocarpus oil or its derivatives?

Generally speaking such questions only arise in lepromatous leprosy. The great majority of neural cases are treated by intradermal injection of ethyl esters and seldom need the intensive treatment laid down for the lepromatous case. The only indication which would point to the necessity for stopping intradermal injection in the neural case would be during the stages of reaction when the lesions are inflamed, angry looking and scaling in such conditions intradermal injections will do more harm than good and in any case the patient's tissues are putting up an effective defence and there is no indication to give further treatment.

In lepromatous leprosy however it is of utmost importance to watch for signs which indicate the necessity for reducing or withholding altogether injections of the hydnocarpus preparations.

Some authorities consider that the sedimentation index test should be relied on to control treatment. Our opinion however is that this is an unreliable test and should only be used in conjunction with clinical observations. It is true that in cases in which caution has to be exercised this index is usually high but because it may not necessarily be so physicians are warned not to put too great a reliance on this test. It can be stated however that a persistently high sedimentation rate indicates some activity either of leprosy or some concomitant condition and that if this is observed then caution must be exercised in pushing the injections.

It is believed that more reliance can be placed on clinical methods for estimating the necessity for regulating treatment and the following clinical tests are of value in the control of treatment.

- 1 Clinical condition of the patients
- 2 Multiple bacteriological examinations
- 3 Temperature

1 Clinical Condition of the Patients

The clinical observation of the patient is by far the most important single method of estimating whether the disease is increasing or whether treatment is lighting up activity. When the patient is being examined prior to injections it cannot be too strongly emphasised that he should be seen in a good light and stripped to the waist. In the case of women clothes should be so arranged that the patient can be examined adequately without embarrassment. This is most important because so often one witnesses the spectacle of a large number of patients being injected without even the most cursory glance at the patient's clinical condition. Once the eye is trained the

dose for children above seven should be 10 minims t d s remembering to start with one drop and gradually increase

When hydnocarpus oil is given by mouth it must be pure injection oil without creosote. While no signs of intolerance have been definitely noticed by us when injections are given hydnocarpus oil by mouth may result in such signs and these are nausea possibly vomiting and diarrhoea. On the appearance of such signs the oil must be stopped and recommenced at the lowest dose after all such signs have disappeared.

Children of nine and above should be encouraged to take injections. Because of the pain caused it probably is wise to commence with subcutaneous injections in children and then go on to the more painful intradermal injection. The maximum dose should be reckoned at 14 c c per every ten pounds of body weight. Assuming a child weighs forty pounds then the maximum dose is 11 c c. Subcutaneous injections are given starting at 1 c c and gradually increasing to 3 c c. When this is reached intradermal injections are commenced the maximum dose of 3 c c of oil is maintained and the ethyl ester mixture started at 1 c c and gradually increased until 3 c c is reached. Generally speaking because of the pain no child should be given more than 3 c c intradermally the balance should be given subcutaneously. Except in dosage above 5 c c half the drug should be given by intradermal injection and half by the subcutaneous route.

Because of the great tendency to natural arrest of the neural lesion only those cases with multiple lesions need receive intensive treatment. The tuberculoid lesions in children unless extensive respond quite readily to intradermal injections. With regard to continuing at school all non infective cases may remain in school provided they are examined every six months in the case of tuberculoid cases and three months in cases which show the incipient lesions of childhood or multiple hypopigmented lesion of simple macular leprosy. In severe infection of nerves in children the great tendency to deformity must be borne in mind but this will be referred to in Chapter XX when the treatment of nerve lesions in leprosy is discussed.

TREATMENT OF LEPROSY—continued

I Control of Treatment

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occurrence of fresh as well as the erythema and spread of existing lesions can be readily noticed. If there is erythema or extension of existing lesions or fresh lesions then caution must be exercised in pushing treatment.

2 Multiple Bacteriological Examinations

It is felt that this method gives a general indication of the progress or retrogression of the disease. In the Lady Willingdon Leprosy Sanatorium all cases are divided into those who are re-examined yearly, six monthly and monthly. While clinical improvement is frequently seen before bacteriological clinical deterioration is preceded by bacteriological deterioration. Therefore if a patient's smears which have been showing gradual but slow improvement deteriorate then this is a warning sign for instance if the result of one examination is as follows

FH	Rt	2+	Ear Rt	1+	Chin	2+	Macules	1	—
	Lt	1+	Ear Lt	2+	Cheek	2+		2	3+
								3	—
								4	—
								5	4+
							Nasal		1+

And after three months the smear results deteriorate thus

FH	Pt	4+	Ear Pt	3+	Cheek	4+	Macules	1	1+
	Lt	4+	Ear Lt	3+	Chin	4+		2	4+
								3	2+
								4	1+
								5	4+
							Nasal		2+

This would indicate that there was a setback in the condition of the patient and that he needed careful observation. More reliance is placed on multiple smears to indicate whether a patient's condition is deteriorating rather than improving for in the latter instance clinical signs precede bacteriological improvement. Again if the bacteriological examination indicates that the patient is not improving then signs of reaction should be searched for and if found the case should be treated along the lines to be laid down. If there is no temperature and there are no definite signs of reaction but smear examination shows deterioration in the results then it is wise to stop subcutaneous injections and continue intradermal injections until the next examination is due.

3 Temperature

The two previous methods of control described are used as a guide to whether hydriocarpus remedies should be pushed and are in our opinion more trustworthy than the sedimentation index test. We have seen at least one instance where the smear results indicated that caution should be exercised and yet the sedimentation index test was under ten and the patient a few weeks later developed acute lepra reaction.

Probably one of the most reliable indices of the retrogression of the disease is seen in the temperature variations between the morning and the evening. If it is possible

all patients should have their temperatures taken during the morning and in the evening between 3 and 5 p.m. If there are marked variations of the temperature whether there is actual fever or not caution should be exercised. The average person has a regular variation between morning and evening temperatures and it is the disturbance of this that is a warning sign. In large institutions the taking of four hourly temperatures is impracticable but if there are suspicions that the disease is increasing in any given case then the patient should be told to see that he gets his temperature taken between 11 p.m. and 1 p.m. in the evening. Whenever the patient's temperature shows an evening rise above 99° F. for more than two consecutive evenings, treatment should be stopped until the temperature returns to normal for the same period.

The decision as to the continuation of treatment or the withholding of treatment presents no difficulty when the patient shows progressive improvement or when there are obvious signs of deterioration. The difficulty comes when consecutive similar results show no improvement or the results show a deterioration in the bacteriological condition. In such cases our practice first is to note whether there is fever. If there are slight evening rises of temperature then all subcutaneous injections are stopped and the patient is placed on intradermal injections alone, reducing the dose to 0.00 cetyl esters mixture intradermally and this is slowly increased once a fortnight or once a month according to the severity of the signs until the temperature shows a rise as usual; this occurs at 3 c.c. This we then consider the reaction level. The patient is then given a rest until his temperature is normal for forty-eight hours and the treatment is recommenced at 0.00 cetyl esters mixture and slowly increased and when 3 c.c. is reached subcutaneous injections are started increasing the dose by 0.00 c.c. every second week until 1 c.c. is reached. The intradermal injections are then raised from 3 c.c. up to 5 c.c. In cases which tend to react it is probably best to give the subcutaneous and intradermal injections on separate days.

If at any point the temperature rises then this is noted and injections are at once stopped and the patient rested until the temperature becomes normal and again intradermal injections are started slowly and increased until 3 c.c. is reached then subcutaneous injections are recommenced and gradually increased to 1 c.c. In this way the dosage is slowly worked up and if the patient is not in chronic reaction with caution the maximum dose of 15 c.c. a week can usually be reached.

The general principle should be to stop subcutaneous rather than intradermal injections and work these up to 3 c.c. before subcutaneous injections are given. Sometimes a patient will not tolerate subcutaneous injections then intradermal injections are cautiously increased to 5 c.c. and should be maintained for three months after which subcutaneous injections can be again started. In this way not infrequently patients who cannot stand moderate dosages are able to reach higher doses and it is thought more effective dosage. Low dosages should be continued so long as there are signs of intolerance to the remedy and often much patience must be exercised. Sometimes the dose remains at 1 c.c. or lower for weeks and if the patient cannot reach the higher dose levels the result of treatment will almost certainly be very poor and the prognosis increasingly grave. If such signs as urticaria or inflammation of any elements of the eye set in during treatment then all injections should be stopped and treatment concentrated on the eye conditions the line of treatment for which will be indicated in the next chapter. The same principle occurs in relation to severe nerve pain. If an acute condition sets in in the course of leprosy then all measures should be taken

to alleviate this condition and special remedies only commenced again after the patient has returned to his normal state of health

II Treatment of Reaction in Leprosy

In Chapter X two conditions have been discussed signs of activity in leprosy and reaction in leprosy which has been further divided into two subdivisions—reaction in neural leprosy and lepra reaction. In the consideration of treatment of reaction this must be viewed in the light of these conditions. In the previous paragraphs general principles of treatment have been discussed and under this head the management and control of active cases has been considered. We now pass on to the treatment of reaction in leprosy and the subject is dealt with under the following heads

A Reaction in neural cases

B Reaction in lepromatous cases

(A) Treatment of Reaction in Neural Cases

This occurs in tuberculoid cases and may show itself in an acute exacerbation of tuberculoid leprosy or a simple nodular case of neural leprosy may develop reaction which shows itself by a change in the clinical condition the lesions suddenly pass into a state of active tissue immunity the result of which is seen in the transformation into tuberculoid leprosy in the active reaction phase. Apart from the occasional bouts of nerve pain and tenderness the pure neural case (neural anaesthetic) does not show any sign of reaction and the question of the treatment of neuritis in leprosy will be considered in a subsequent chapter

Treatment of Reaction in Tuberculoid Leprosy

This invariably is a manifestation of acute tissue immunity (? allergic) and the signs of this condition have been described. When the reaction is mild it does not need special attention except that it must be remembered that intradermal remedies must be withheld so long as the lesions are marked by erythema scaling or ulceration. Unfortunately this condition is sometimes associated with tingling and burning of the patches and continuous fever accompanied by nerve pain. The burning and tingling of the body with acute tenderness of the patches is difficult to treat. Anodynes such as aspirin caffeine and phenacetin should be tried and sodium salicylate with sodium bicarbonate should be given. If sleeplessness is troublesome the following prescription may be found useful

R Aspirin	grs V	(0.3 grm)
Codeine	gr $\frac{1}{2}$	(0.016 grm)
Phenacetin	grs V	(0.3 grm)

This should be administered when the pain is severe and the last thing at night preceded by a dose of a sedative mixture such as

R Chloral hydrate	grs XX	(1.0 grm)
Pot bromide	grs V	(0.6 grm)
Sodium bromide	grs X	(0.6 grm)
Aquam ad	$\bar{3}$ $\frac{1}{2}$	(15.0 ml)

When the pain and discomfort is not relieved by aspirin and phenacetin and is very distressing, the following sometimes succeeds

R Medinal	grs VII (0.45 grm)
Pyramidon	grs IV (0.25 grm)

This is given last thing at night preceded by a bromide and chloral mixture If in spite



FIG 115—5 cc subcutaneous and intramuscular injections of 1% solution of novocaine in the arm and leg to relieve the pain and discomfort of the disease. The patient is given 10 cc of novocaine in the arm and leg.



FIG 116—9 cc subcutaneous and intramuscular injections of 1% solution of novocaine in the arm and leg.

of this the patient is sleepless then the following may be given at night and the medinal and pyramidon powders during the day

R Chloral hydrate	grs XX (1.0 grm)
Pot bromide	grs XX (1.0 grm)
Tr opii	℥ XX (1.0 ml)
Aquam ad	℥ I (0.0 ml)

One ounce is given at night and must not be given for more than three consecutive nights. This remedy usually ensures at least three nights good sleep and the patient then will respond more readily to the less powerful drugs. Occasionally when the condition is very distressing or in instances of severe pain when it is inadvisable to give morphine injections the following powder may be given with bromide at night

R Pyramidon	grs IV (0.25 grm)
Medinal	grs VII (0.45 grm)
Heroin	gr ½ (0.01 grm)

It should be remembered that such a powder must only be used when all else fails and not continuously. When the skin is highly sensitive to the slightest touch especially

when there is scaling or ulceration of the lesions then the bed clothes should be supported on a cradle and prevented from contact with the acutely sensitive skin

When as sometimes is the case there is active ulceration and excessive crust formation the patient should be placed in a hot saline bath the crusts softened and then gently removed with olive oil or other bland oil. No force must be used or there will be considerable bleeding. The ulcerated areas are not bandaged but covered with plain hydnocarpus oil and the patient placed under a cradle. If there is a great deal of crust formation which does not readily separate on soaking Lassar's paste will help in the separation of the scales especially on the arms and legs.

R	Acid salicyl	5	I	(4.0 grm)
	Zinc oxide	5	II	(8.0 grm)
	Amylum	5	II	(8.0 grm)
	Vaseline	5	IV	(16.0 grm)

This should be applied as a thick paste on gauze or lint and the limbs bandaged. In a few days the crusts will separate and leave raw surfaces. These should be dressed with plain hydnocarpus oil or the following ointment.

Chronic Ointment

R	Acid boric	grs	XX	(1.0 grm)
	Oil eucalyptus	ml	XX	(1.0 ml)
	Bis subnit	5	I	(4.0 grm)
	Zinc oxide	5	III	(12.0 grm)
	Castor oil	5	I	(30.0 ml)

Instead of the above the following ointment as an alternative may be found more useful.

R	Oil hydnocarpus	5	III	(12.0 ml)
	Oil eucalyptus	5	II	(8.0 ml)
	Zinc oxide	grs	LX	(4.0 grm)
	Dettol	5	I	(2.0 ml)
	Vaseline	5	I	(30.0 grm)

If the ulcerated surface becomes secondarily infected and the fever is considered to be due to this then the sulphonamide products may be tried. Not infrequently this condition in its more severe forms is associated with pyrexia and in such cases a course of mercurochrome is sometimes beneficial. Five cc of a 1 per cent solution intravenously every third day may bring the temperature down six to eight injections should be given. If the temperature returns to normal for forty eight hours the injections are stopped but if fever persists after six to eight injections there is no point in continuing the injections.

The more severe conditions of reaction are usually seen in the atypical border line or intermediate cases and sometimes the reaction condition continues for six or seven months accompanied by much emaciation. If the fever which is probably that of the hectic type continues for more than three months then the case is usually one belonging to the atypical group and this can usually be confirmed by biopsy and by the fact that the lepromin test is not infrequently negative.

The management of such cases needs great patience, care and nursing skill but they invariably recover leaving a considerable amount of deformity and scarring. Once a diagnosis of acute reaction in tuberculoid leprosy or in the atypical form is made the patient should be encouraged by the fact that he will recover. If such a

condition as described lasts more than seven months then one's diagnosis as to type must be seriously reconsidered for even the worst reactions in this type have shown recovery and healing within seven months. It is most important to differentiate this condition from lepra reaction for its immediate prognosis is invariably good although deformity sometimes disabling deformity may result whereas as will be pointed out the ultimate prognosis in lepra reaction is poor.

(B) Reaction in lepromatous cases

True lepra reaction as stated previously only occurs in lepromatous leprosy and may be acute sub acute or chronic. Acute reaction may pass into the sub acute or

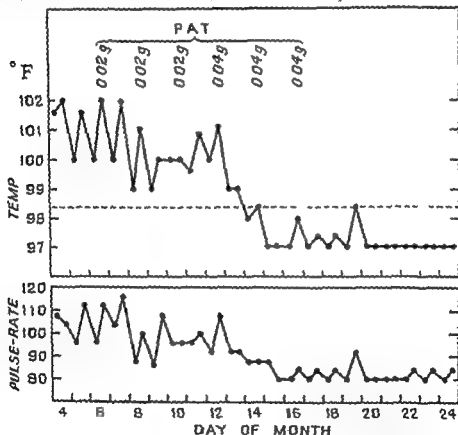


FIG. 11.—Temperature falling to normal and pulse rate falling to normal by treatment with PAT.

chronic stages but the majority of acute reactions respond to treatment. The sub acute and chronic stages are more insidious in their onset and more difficult to control.

(c) Treatment of Acute Lepra Reaction

The symptoms and signs have already been described but the warning must again be reiterated that a patient who has leprosy and becomes febrile may be suffering from

some complicating condition and all measures must be adopted to exclude such a possibility before special treatment for lepra reaction is commenced. Therefore the first step in its treatment is to treat the patient along the usual lines of a non specific febrile condition.

The patient is put to bed and given a powder containing aspirin grs 4 calomel grs 2 and Dover's powder grs 4. This is followed the next morning by a saline purge.

It is remarkable how frequently fever drops after the bowels have been well opened.

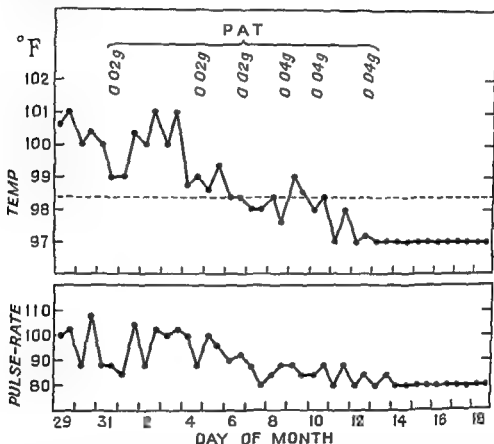


FIG. 118.—Temperature chart illustrating the control of lepra reaction by intravenous injection of I.A.T.

and the patient has perspired. If the fever continues the patient is placed on an alkaline mixture containing sodium bicarbonate grs 30 to the ounce given every four hours. Sodium salicylate is added to the mixture if the joint and bone pains are troublesome. Miltalba is given every morning. For pain a tablet of combral and if insomnia is troublesome a bromide mixture may be given. If combral is too expensive a powder containing aspirin grs 4, phenacetin grs 4 and caffeine grs 2 may be prescribed. This will frequently relieve pain and thus remove the greatest cause of sleeplessness. If however the latter is troublesome codeine grs 1 may replace caffeine and the powder given at night followed by a bromide mixture. Proprietary remedies such as Codiparin

or Vegamin will be found useful. The diet should be light with plenty of fluids glucose being added if thought necessary.

If the temperature does not come down within three or four days then one of the antimony products is indicated. The one which has been in vogue is potassium antimony tartarate (P A T) but it needs special care in its administration owing to the fact that

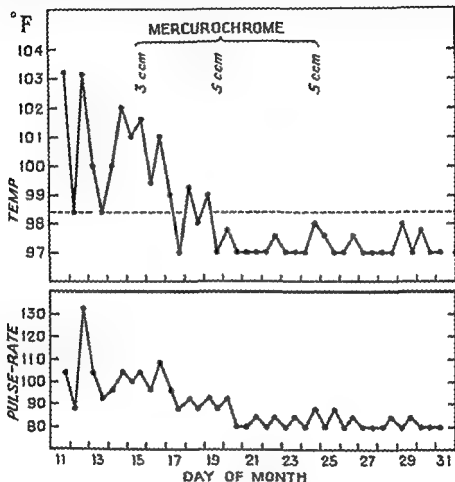


FIG. 110.—Temperature and pulse rate of a patient treated with Mercurochrome in the treatment of leprosy.

it must be given intravenously and unless given carefully will produce sloughing of the tissues. The dose is 0.02 grms in 2 c.c. saline intravenously every other day for three doses then 0.04 grms every other day for three doses. Where it is difficult to administer remedies intravenously or in institutions where medical aid is not immediately available then the pentavalent preparation Fantom (Chico & Co.) may be given. The dose is 1 c.c. intramuscularly every other day for six doses. If the temperature does not respond and one still feels that the condition may be due to acute lepra reaction one is then justified

in giving a course of P A T 1 c 0 04 grms intravenously every other day for six doses. Sometimes although we think rarely P A T acts when Fintorin fails. If the temperature does not come down with Fantorm we believe that the diagnosis should be reconsidered this becomes all the more essential if P A T also fails to bring the temperature down. In fact we teach that if the antimony products when properly used fail to bring the temperature down either the physician is not dealing with acute lepra reaction or it is complicated by some other condition.

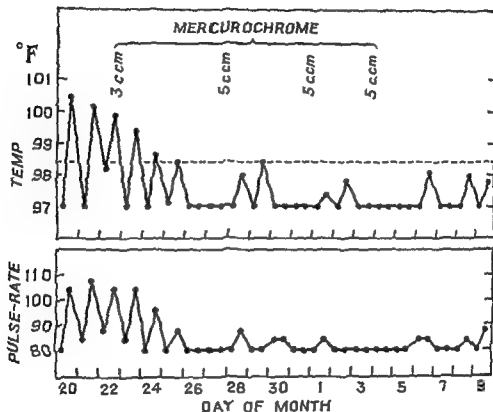


FIG 100—Temp rature chart illust at ing the control of fever by intravenous injection of mercurochrome in sub acute lepra reaction

(b) Treatment of Sub acute Reaction

As stated acute lepra reaction usually responds to treatment but when a patient suffers from a number of attacks the intervals between the attacks tend to become shorter and he is liable to pass into the sub acute stage. The general lines of treatment are similar to those indicated for lepra reaction the distressing sequela of nerve pain will be considered later. The fever in sub acute reaction is more difficult to deal with and the drug which has been found more generally useful is mercurochrome in a 1 per cent solution the dose is 5 c c for those above fifteen and 3 c c for children. It is important to administer chemically pure mercurochrome for some commercial products are not for internal medication and if given cause serious febrile reactions. Therefore mercurochrome for intravenous injections should always be ordered. It is given intravenously every third

day Occasionally when mercurochrome fails then fluorescin may be tried this is given in a 2 per cent solution in 2 per cent sodium bicarbonate intravenously every third day Great caution must be used in the administration of fluorescin and it should be given in between meals and the patient kept in a room in a dull light and not exposed to direct rays of the sun for twenty four hours after the injection Not more than six injections should be given These measures not infrequently control the fever and with the general measures employed will bring the reaction stage to an end but in advanced

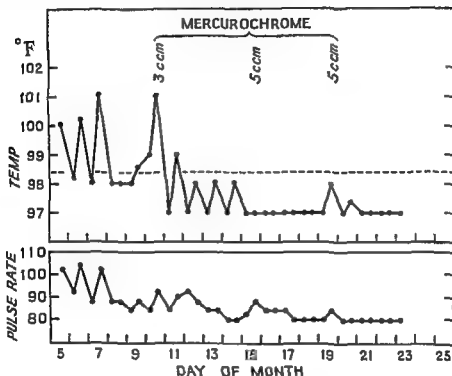


FIG 11—Temperature chart illustrating the effect of off by intravenous injection of mercurochrome in a patient with leprosy

lepromatous cases this usually is only temporary to be followed in another few weeks or even days by a further rise of temperature and a gradual development of chronic reaction

(c) Treatment of Chronic Reaction

This can be considered under general treatment and treatment of special complications e.g. breakdown of nodules giving rise to ulceration The general treatment is mainly symptomatic and prescriptions and guidance have already been laid down with reference to this If the fever does not subside after the administration of the various remedies advised in acute or subacute reactions then there is little value in pressing the treatment and the condition must be treated symptomatically until the temperature comes down This condition in such instances may continue for many months and the

patient pass into a weakened cachectic condition and die as a result of the prolonged reaction or fall prey to one or other of the more serious complicating conditions e.g. dysentery, tuberculosis, nephritis.

As a result of the almost continuous bouts of fever patients may become anaemic and this should be combated by adequate doses of iron. Vitamin B complex may be given either as yeast (11 drms. four to six times a day) or if the person can afford it one of the reputable preparations of concentrated vitamin tablets which are on the market. If Marmite is used adequate doses ($\frac{1}{2}$ to 1 oz.) should be given.

In the stage of chronic reaction nodules tend to break down and ulcerate. If the ulcers are dirty then they should be cleaned with eusol dressings or with saturated mag. sulph. compresses followed by eusol dressings. When the discharge has diminished and the ulcers are clean one of the two ointments recommended or the following may be given.

R	Ol eucalyptus	3	IV	(15.0 ml)
	Honey		II	(56.7 grm)
	Cod liver oil		II	(56.7 grm)
	Zinc oxide	1	I	(28.4 grm)
	Bis. subnit.		II	(56.7 grm)
	Vaseline		III	(85.1 grm)

If the nodules have not ulcerated then frequent application of a carbon dioxide snow will be found useful. This must be applied with firm pressure from forty seconds to one minute according to the consistency of the nodule. The more fibrous ones may need the longer time. Under constant application a crust forms over the nodule which ultimately separates and the nodule frequently then dries up and gradually disappears. Applications should generally not be repeated until crusts have separated.

The one remedy which seems of value in chronic reaction with breaking down and ulcerating nodules is the salts of gold. Solganol B oleosum has been recommended but Myocrisin only (M & B) may be used instead. In all such cases where other remedies have failed a course of gold may produce healing of the ulcers. The points to remember are (1) that the dosages must be low; (2) a second course should not be given in less than three months; (3) the drug is given intramuscularly once a week in the following dosages:

Three injections of 0.01 grm
Three injections of 0.05 grm
One injection of 0.10 grm

The treatment of chronic reaction is not satisfactory but one usually can make the patient's life at least bearable and tide over the more distressing symptoms for a considerable period but it must be borne in mind that this condition presents the most acute nursing problems and all the care, patience and fortitude of the medical staff are needed to maintain the morale of the patient when he gets to this condition. The newer drugs such as promin, diasone, promizole appear to be encouraging when used in this condition but these are still in the experimental stage and must be given over a long period.

TREATMENT OF NEURITIS IN LEPROSY

NEURITIS in leprosy is usually a reaction condition but has not been described under lepra reaction because its relief is of such importance in maintaining the well being of the patient that special consideration must be given to the management of a case complicated by neuritis. In the chapter on Pathology it was pointed out that nerve tissue reacts defensively to the attack by *M. leprae* in two different ways. In one form there is a violent tissue response as seen in tuberculoid leprosy; in the other there is an interstitial fibrosis which results in fibrous tissue formation which contracts and ultimately strangles the nerve fibres. The ineffectiveness of certain treatments in leprosy complicated by neuritis we feel has been because the physician has not always realised the essential difference in the pathology of these two types of neural lesions.

Neuritis due to Active Tissue Immunity

This type of case is always seen in tuberculoid leprosy and more rarely in some of the atypical lesions. As has been described granulomatous masses form actually in the nerve tissue which is invaded by round cells, epithelioid cells and giant cells. Owing to the pressure of the granuloma on the nerve sheath abscess formation is likely to occur if the condition is not relieved. Neuritis in tuberculoid leprosy and to a less extent in the atypical forms shows itself in two ways.

- (a) Acute (b) Chronic

(a) Acute Neuritis

This invariably occurs in the reacting case and all signs of active tissue defence as described in the previous chapter are present. The affected nerves are swollen grossly enlarged and acutely tender. Not infrequently there are signs of abscess formation. The swelling of the nerve may not be uniform. It is a safe assumption to conclude that in all cases which present signs of marked tissue immunity in the skin when they manifest symptoms of reaction in the nerves that the reaction is of the same nature. The only method of treatment in these cases is operative and the longer this is delayed the more likely are abscesses to form and secondary fibrosis result. Therefore the earlier the operation is undertaken the better the result. The object of the operation is to dissect away the constricting outer nerve sheath and so allow for the free expansion of the inflammatory exudate and thus relieve the pressure and if the operation is undertaken in the early stage there is little likelihood of secondary fibrosis. When the tuberculoid condition subsides there is a fair chance of regeneration of the nerve fibres and recovery of function. In one case in our experience the whole of the ulnar nerve was scraped away but after six months of massage and passive treatment complete restoration of function was achieved.

Where a diagnosis of acute tuberculoid reaction in a nerve is made especially

when it involves the ulnar nerve operative procedures must be undertaken without delay. Several operative procedures have been advocated

- (1) Nerve stretching
- (2) Incision of sheath and stretching of nerve
- (3) Excision of the sheath over the whole length of the affected portion

Nerve stretching by itself is not to be recommended and is a useless procedure. The second procedure is not sufficiently radical and therefore the third method advocated by Lowe and the Calcutta School is the method of choice.

Description of Operation. This is usually only indicated in acute tuberculoid reaction in the ulnar nerve. It must be borne in mind contrary to the generally accepted opinion that the ulnar nerve is affected chiefly in the middle two thirds of its length and not at the bend of the elbow. Because it is impossible adequately to anaesthetise an inflamed nerve a general anaesthetic should be given. A tourniquet may be used for the initial step for the removal of the nerve sheath can then be effected without the operation field being obscured by blood. A tourniquet is not absolutely essential but is more convenient for the oozing from small vessels may be troublesome and unless haemostasis is effective the operation is more difficult. We have seen no untoward result from the application of a tourniquet. The nerve is exposed for four to six inches at its points of greatest thickness—in the centre of the arm and not at the elbow—as it passes on the inner side of the artery on the intramuscular septum. The nerve is freed from its bed and the sheath incised along the whole length of the exposed nerve. An aneurysm needle is passed round the sheath behind the nerve and the whole sheath freed at the same time stretching the nerve. The sheath then is completely removed by cutting it away with a pair of fine scissors. If an abscess is discovered the procedure is the same but the abscess is carefully scraped. If a tourniquet has been applied it is now loosened bleeding points cauterised and the nerve replaced in its original position and the wound sutured without drainage. The relief is immediate and there are seldom operative complications unless an abscess has not been thoroughly dealt with or the nerve sheath not completely removed. When the wound will not heal owing to fresh abscess formation then the wound must be reopened and a more thorough removal of the nerve sheath with evacuation of the abscess completed. After treatment in the way of exercise and massage will be described under the treatment for other forms of neuritis. Generally speaking operative procedure is only of value in the case of the ulnar nerve and while it will relieve pain it seldom prevents the onset of or recovery from drop foot. If the great auricular nerve is grossly involved as this is solely a cutaneous nerve of minor importance the whole length of it from its appearance at the sterno mastoid muscle to the auricle can be excised. Care must be taken thoroughly to excise the whole length of nerve for unless it is efficiently done an abscess at the edge of the muscle will result. The operation cures the pain and removes an unsightly nerve. Enlargement and abscess formation of superficial cutaneous nerves are of little importance. If these are painful and unsightly the whole of the grossly swollen portion may be excised. In cases of motor and cutaneous nerves combined complete excision of the nerve must never be undertaken.

(b) Chronic Neuritis

If neuritis in tuberculoid leprosy is not adequately treated a nerve abscess may ultimately break down through the skin and result in a sinus. This sinus will continue

until the necrotic material has been completely extruded or until an operation has been undertaken to deal with the abscess along the lines already indicated. If however such a condition is allowed to arise the chances of permanent damage are considerable for secondary fibrosis is likely to set in. It is therefore better at any stage to scrape the abscess and remove the sheath even though this means complete loss of function for when the necrotic material has been evacuated and only a few fibrous strands remain after the operation yet I have seen regeneration of the nerve and recovery of function.

In neural anaesthetic leprosy the tissue reaction is one of interstitial fibrosis. This results as explained in the destruction of the bacilli and ultimate strangulation of the nerve fibres and loss of function. The treatment of this condition is similar to that which is about to be described under neuritis in lepromatous leprosy.

Neuritis in Lepromatous Leprosy

Neural anaesthetic leprosy is the only type of leprosy in which nerve tissue alone is affected. In all other forms of leprosy there are cutaneous lesions as well as neural hence the old term cutaneous or skin leprosy gives an entirely wrong conception of what is known as lepromatous leprosy. Similarly while lepromatous leprosy does not necessarily show neural signs yet nerves are affected. If for instance the ulnar or peroneal nerve is examined and smears taken from the nerve sheath *M. leprae* can be discovered almost invariably. In sections if the correct technique is used for staining for acid fast bacilli bacilli can be demonstrated in the nerve. As has been mentioned even though bacilli can be demonstrated in the nerve sheath no tissue reaction is noticed hence the nerves usually stand out clearly in the corium in sections from lepromatous leprosy. The changes noted may be a slight proliferation of the perineurium and some swelling and oedema of the individual nerve bundles. While this applies to early leprosy as the condition advances the bacilli lying between the individual nerve bundles act as foreign bodies and an interstitial fibrosis is which in contrast to that of neural anaesthetic leprosy sets in slowly but nevertheless the end result is the same namely destruction of the nerve and complete loss of function. Neuritis in lepromatous leprosy which is always of the nature of an interstitial fibrosis is seen in three forms

- (a) Acute (b) Sub acute (c) Chronic

Before describing the above three conditions it might be well to mention that in all forms of interstitial fibrosis operation is contra indicated for unlike neuritis in tuberculoid leprosy where the granulation tissue is within the nerve pressing on the sheath from within there is no granulation tissue within the nerve but a fibrosis between the individual nerve bundles and hence any operative interference will only tend to increase the fibrosis and the last stage is liable to be worse than the first.

(a) Acute Neuritis

This is chiefly seen in lepromatous leprosy but may occur in the simple macular or neural anaesthetic case. The onset is sudden it may be precipitated by injudicious use of remedies such as iodides and the following signs and symptoms are prominent. There is oedema and tenderness of the nerve without actual abscess formation the nerve is usually thickened swollen and oedematous and very tender to the touch. It is in just this type of case that great gentleness needs to be observed in examination and palpation of the nerve for much pain can be caused by forgetting that nerves in lepromatous

leprosy can be enlarged and tender. It is to be remembered that in neural anaesthetic leprosy the same type of lesion may be present for the main brunt of the attack is on the large nerves and these may become swollen tender and even oedematous with all the signs of an acute condition. Although neuritis in neural anaesthetic leprosy is more likely to be subacute and chronic acute neuritis in lepromatous leprosy or more rarely in the neural anaesthetic or simple macular type may cause great pain and the symptoms are sometimes so acute that the condition demands measures for immediate relief. The pain frequently is so severe and the remedies recommended for other types of neuritis take so long to bring relief that the patient's acute distress must be relieved. Three procedures may then be adopted:

- 1 Alcohol injections into the nerve sheath
- 2 Deep X ray treatment
- 3 Administration of adrenalin or ephedrine

1 *Alcohol Injections into the Nerve sheath* In the first type when the nerve sheath is oedematous and swollen and the case is one of lepromatous leprosy where there is no danger of abscess formation an injection of alcohol into the nerve sometimes produces lasting relief. It will always bring immediate if temporary relief and is worthy of a trial. The technique is as follows using a 2 c.c. syringe with a small needle (size 15) palpate the enlarged nerve and then run the needle of the syringe along the palpating index finger and as soon as the nerve is felt under the needle give a sharp poke and enter the nerve sheath. 3-5 of a 75 per cent solution of alcohol is injected directly into the nerve sheath. The pain momentarily is excruciating but the relief if the injection is given properly is instantaneous. If it is impossible to penetrate the nerve sheath a little novocaine (2 per cent) may be injected around the nerve and then without withdrawing the needle recharge the syringe with alcohol and inject this around the nerve. This method is less effective but gives temporary relief and certainly reduces the pain considerably.

2 *Deep X ray Treatment* Some years ago Expen (1940) advocated deep X ray exposures for acute neuritis and in the stage of oedema where fibrotic tissue formation is not marked dramatic results have been noted. Four to six exposures are usually necessary and if no relief is noted at the end of this period deep X ray should be abandoned. In all cases of acute neuritis in the course of lepromatous leprosy especially if associated with fever a course of potassium antimony tartrate (I A T) along with general measures for the relief of nerve pain is worthy of consideration for when lepra reaction subsides the neuritis frequently is greatly relieved.

3 *Ephedrine or Adrenalin* Injections of adrenalin 10 in 30 saline have been advocated for painful neuritis. These sometimes help and should either be given subcutaneously or along the course of the nerve. Ephedrine has been recommended in place of adrenalin but is not so effective and it is doubtful whether it is of much value by mouth. Whenever there is acute pain local applications e.g. recthyol and glycerine or antiphlogistine are of value and the limb should be splinted. The patient frequently needs to be put in bed and the preliminary treatment indicated for lepra reaction should be given. It has not infrequently been noted that acute reaction in lepromatous cases is accompanied by nerve pain this sometimes is relieved by P A T and therefore should be given when the symptoms suggest a diagnosis of acute lepra reaction. Diathermy has been found useful and is worth a trial.

(b) *Sub acute Neuritis*

This complication is usually seen in the form of a multiple neuritis. The pain is not so severe as in acute neuritis but the patient is conscious of pain and any manipulation of the nerve causes distress as the nerve is tender. The remedies described for acute neuritis may be tried the patient's general condition should be treated and such irregularities as constipation dealt with. It is in this condition and in the chronic condition that dietetic treatment seems of more value. The diet advocated is a whole wheat diet and the method adopted is described below.

(c) *Chronic Neuritis*

This is the final result of the interstitial fibrosis seen sometimes in neural but more often in advanced lepromatous leprosy. This is a distressing condition for the patient is always conscious of pain and frequently associated with this condition is found severe bone pain. This is elicited by pinching the clavicles or lightly pressing the anterior surfaces of the tibia or the radius and ulna. As this is a chronic process some of the remedies described for the relief of the more acute conditions are of little avail. Anodynes such as phenacetin, caffeine and aspirin are given. If these fail and the pain is very severe the powder containing pyramidon and medinal may be prescribed. Combined with such a measure local applications such as antiphlogistine or ichthyol and glycerine may be tried and the arm put in a sling or splinted. Opium or morphia should never be administered either by mouth or in the form of an injection except occasionally in the form of a hypnosis if the patient has had many sleepless nights then one dose of the bromide chloral tropon mixture may be given at night to ensure sleep. On no account must injections of morphia or heroin be prescribed because this condition is chronic and a morphia habit is in danger of being acquired if injections are ordered. The bowels are kept open by saline or other suitable purges and if there is evidence of lepra reaction this is dealt with in the manner previously described. Where the pain is severe none of the methods are of any permanent value. It has been found that this condition may respond sometimes dramatically to a whole wheat diet. Results cannot be expected immediately but within three months in a series of cases 80 per cent had complete relief of bone pain and 69 per cent of nerve pain the rest had partial relief. The total number of cases was fifty even. The method of administration is as follows.

All rice is eliminated from the standard diets of the Sanatorium and instead whole wheat was taken. This was lightly ground on the curry stone and cooked in the same way as rice and distributed to the patients who ate it along with their usual curries. In the evening chapatties¹ were eaten. For adults 21 oz of wheat per diem were supplied and for boys 12 oz. Wheat prepared in this way needs slightly longer cooking than rice and extra salt should be added.

From time to time other methods of relief of nerve pain have been advocated. Gass (1938) tried cobra venom and claimed a 75 per cent relief of pain and said that it had given far better results than anything else. The recommended dosage was as follows: first day 0.1 cc third day 0.2 cc fifth day 0.3 cc increasing thus until a dose of 1 cc is reached. We failed to confirm that this remedy was of real value.

¹ A chapatti is a type of pan-bread made with whole flour and is served by order in India especially in the north.

but possibly it is of most benefit in the arrested case where the chief symptom is intense burning and pricking pain.

Diphtheria formal toxoid 0.5 c.c. to 2 c.c. once a fortnight has been advocated but this method is not recommended for it gives little or no relief and is liable to set up reactions in lepromatous cases. Intradermal injections along the course of the nerve have been advocated by Chatterjee but we have never experienced any appreciable relief by this method and fail to see the reason for recommending this procedure. For the associated bone pain deep X rays have been tried with some success but it will not appreciably affect the chronic nerve pain for this is only of value in the swollen and tender painful phase. It should be remembered that in every case of neuritis in leprosy the patient should be warned of the possibility of deformity. This is especially necessary in the chronic interstitial variety. Nevertheless even when deformity has set in much alleviation can be obtained by appropriate treatment. Muscular atrophy in leprosy is due to two factors. (1) loss of function as a result of damage to the nerve supply to the muscle (2) disuse atrophy. While the former is difficult to prevent the latter need not occur. In all cases after the acute condition has passed massage and appropriate active and passive exercises should be prescribed. In addition electrical stimulation of the muscles should be undertaken. This is best done by the faradic current but the toy hand battery in which the current is generated through an electric magnet by turning a handle should not be despised. If a patient is willing to spend the time taken to massage his hands exercise his affected muscle and persistently apply some kind of electrical stimulus to the limb much deformity can be prevented and disuse atrophy and the worst effects of claw hand avoided. Drop foot and facial paralysis very seldom recover even with the most vigorous measures. All cases of neuritis in leprosy of the interstitial variety should be placed on a whole wheat diet. While injections or tablets of Berin or other vitamin B₁ preparations may be prescribed we have never been convinced of their usefulness and consider that the whole B complex should be advised rather than one of its several components.

In conclusion it may be said that much can be done for nerve pain in leprosy and it is our confident hope that by far the great majority of patients complaining of nerve pain will be helped by one or other of the methods which have been outlined even though complete relief is not always possible of achievement in every case.

Physiotherapy Actinotherapy and Electrotherapy in Leprosy¹

In all general hospitals electrical treatments and massage are considered essential measures in dealing with the results of nerve lesions yet the number of hospitals or sanatoria dealing with leprosy which can boast of a department devoted to work of this kind is comparatively small despite the prevalence of neural involvement in leprosy.

It is not our purpose to deal with technical details on a subject of such magnitude but to concentrate on practical issues and so it may be worth while to begin with a brief description of the various types of treatment which should be undertaken in the electrical department of a leprosy institution and then give an account of the application of the treatments to leprosy lesions.

¹ Dr Donald Dow lately Superintendent of the Leprosy Hospital, Diphtheria, Nazam's D. M. N. 18, has kindly contributed the following account of simple physiotherapeutic methods which he has found of value in the treatment of nerve lesions in leprosy.

Electricity

The following are the procedures in common use

Diathermy This is a high frequency alternating current with oscillations too rapid to allow of chemical changes in the conductor through which it passes so a great thermal effect can be produced. The current flows chiefly round a limb to which the electrodes have been applied in the plane of the subcutaneous or deep fascia and little passes directly through the muscles in a direct line from one electrode to the other. Short wave therapy on the other hand produces heat in the depth of the tissues. This is a high frequency current with oscillations of 10 to 100 million cycles per second.

Galvanism This is a constant current which is used along with the faradic current in the testing of muscles and nerves. It stimulates muscles into contraction at the moment of making and breaking of the circuit of the current. It also increases cell activity in the tissues through which it passes.

Faradism This is an alternating current with power to stimulate nerve fibre.

Sinusoidal current is an alternating current with the current rising from zero to maximum then falling to zero to be followed by the same process but with the current flowing in the opposite direction.

Light

There is no need for an elaborate description of the various types of lamps for the production of ultra violet or infra red light.

Ultra violet light is used for its general tonic action and confers increased resistance to infectious ailments. It is supposed to produce vitamin A in the deeper layers of the skin.

Infra red light is one of the commonly used sources of dry heat which produces vasodilatation of the surface vessels leading to an increased flow of arterial blood.

Massage

The manipulations chiefly used are

- (a) Effleurage—a stroking movement
- (b) Petrissage—the muscles are drawn away from the bone and squeezed
- (c) Kneadings—the muscles are moved in a circular manner by the hand of the operator
- (d) Vibrations—a sensation of vibration is transmitted through the hand of the operator to the patient
- (e) Frictions—small circular movements usually given around joints or along the course of nerves
- (f) Tapotement—percussive movements

In all the manipulations pressure is always given in a centripetal direction thus greatly improving circulation assisting in the absorption of inflammatory exudates maintaining muscle tone and preventing adhesions.

Active passive and resistive movements are performed to increase mobility and strengthen muscles. Patients should be encouraged to perform simple exercises to strengthen or re-educate muscles.

X rays

A small X ray plant is very useful for the detection of bone lesions.

Treatment of Lesions

Nerve involvement with its attendant pain or deformity is extremely common in leprosy and consequently cases of that type form the bulk of the patients in the electrical department

Unless in acute cases where the relief of symptoms is urgent it is advisable to test the electrical reactions of the muscles. Normal muscles react briskly to the faradic current through stimulation of the nerve fibres. The galvanic current causes direct stimulation of the muscles, the response taking place at the make and break but the contraction around the cathode is greater than that at the anode i.e. $KCC > ACC$. In muscles to which the nerve supply has been cut off by disease there is no response to stimulation by faradism and the response to galvanism is weak and torpid but persists while any muscle cells exist. The making of the current however produces a greater effect than the breaking i.e. $ACC > KCC$. These phenomena constitute the reaction of degeneration and are a sign of complete nerve destruction. It follows therefore that treatment cannot produce benefit in lesions due to destruction of nerves in leprosy when muscle testing reveals reactive degeneration.

It is in the treatment of pain due to swelling and tenderness in the larger nerves (ulnar, peroneal etc.) that electrical treatment is of most value.

Routine treatment should be given daily and consist of

- (a) Fifteen minutes massage to affected limb
- (b) Fifteen minutes interrupted galvano faradism in the form of electric bath
- (c) Twenty minutes hydro diathermy—about 125 amp of current
- (d) Simple exercises to encourage use of affected muscles

This treatment is generally beneficial in cases with tender nerves whether they can be classified as neural or lepromatous and the best results are naturally to be expected where the condition is not of long standing. In neural cases when there is no reaction of degeneration and where the muscle tissue has not been replaced by fibrous tissue there is often marked improvement in deformity and sensation. It is advisable to give hydro diathermy rather than direct application of electrodes in these cases unless expert observation is available all the time for the great danger is burns on anaesthetic areas.

For acute pain whether the nerves are thickened or not we have not found diathermy of great benefit till the acute phase has subsided. Exposure of the tender area to infra red light seems to bring relief in these cases and when the acute pain has subsided diathermy with massage is indicated followed later by electrical treatment. This holds true of the very tender nerves in lepra reaction and also in those found in the reactive phase of tuberculoid leprosy though in some of these we have found that after exposure to infra red light operation was necessary and after the wound had healed treatment was continued with diathermy, massage and finally electricity.

In cases of chronic nerve pain when the response to other forms of treatment has not resulted in benefit the sinusoidal current is often successful and should always be tried for the low grade pain attendant on chronic nerve involvement often makes the patient feel very miserable. In old standing cases with marked muscle wasting as a result of nerve destruction little benefit can be expected from electricity.

The use of ultra violet light has no special place in the treatment of leprosy. It

has no effect on skin lesions generally and in some cases of lepra reaction seems to aggravate cutaneous lesions

Infra red radiations on the other hand are a very valuable aid in several conditions e.g.

Trophic Ulcers : Where conservative treatment has been decided on 20-30 minutes exposure to infra red light often produces very pleasing results when other types of treatment have failed

Local reactions following injections : Many of these cases go on to abscess formation but if treated in the early stages with infra red light the swelling subsides and operative measures are unnecessary

Neuritis : In acute cases before it is advisable to give diathermy and electrical treatment infra red radiation should be given and is very comforting to the patient

The one great use for X rays in leprosy is for the examination of bone conditions. The rarefaction and absorption of bones can be demonstrated and it is also valuable to find out in cases of trophic ulceration with mixed infection if there is necrosis of underlying bone before deciding on the extent of operative interference required

TREATMENT OF EYE NOSE AND THROAT LESIONS

A TREATMENT OF LESIONS OF THE EYE

The danger of leprosy of the eye has been stressed already and therefore it cannot be too strongly emphasised that any affection of the eye due to leprosy must be taken seriously and treated adequately. The lesions of the eye have been divided into those resulting from actual invasion by *M. leprae* and those due to mechanical causes through damage to the nerve supply.

Treatment of Eye Lesions in Neural Leprosy

The first principle is to protect the eye—this is best done by putting liquid paraffin or castor oil drops in the eye especially at night. If there is an associated conjunctivitis the eye should be cleaned before the drops are inserted by washing with the following lotion

Boric acid	grs	℥	(0.6 grm.)
Zinc sulphate	grs	℥	(0.03 grm.)
Aquum ad	℥	I	(30.0 ml.)

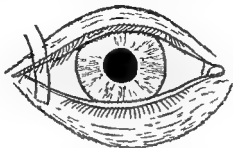
If there is paralysis resulting in lagophthalmos and the cornea is exposed when the eyes are closed then the operation of tarsorrhaphy should be done.

There are two methods of operation—both involve creating raw surfaces on the upper and lower lids and approximating these surfaces. The first method has been described by Cass as follows:

Iodine is painted over the skin surface of the lower lid which is to be excised and then the eye is washed thoroughly with 1:5000 corrosive sublimate solution. Two drops of 4 per cent cocaine solution are instilled in the eye three times at five minute intervals. One per cent novocaine solution is used as infiltration anaesthesia for the skin and subconjunctival surface and a few drops of 1:1000 adrenalin are added to the novocaine solution to control excessive bleeding. The upper lid is everted and novocaine solution injected into the portion of conjunctiva to be excised—an amount sufficient to produce moderate ballooning of the tissue is used. The skin area of the lower lid is dealt with in a similar fashion.

Operation. Before beginning the operation it is advisable to determine where the new lateral canthus is to be. This point is marked on the upper and lower lids. The upper lid is now everted and an incision 5 mm. deep is made along the intermarginal line and carried out to the lateral canthus. Then an incision sufficiently deep to excise the conjunctiva is carried upwards for about 5 mm. after which it is carried out to the lateral canthus—the incision being in the form of a triangle with its base towards the medial parts of the eye and its apex at the lateral canthus. A similar incision is made over the skin of the lower lid as shown in the diagram. It is very important at this time to remove the lash bearing area of the lower lid which is to be included in the suture. If this procedure is not thorough the result is that lashes grow again with much

discomfort to the patient. The union of the two corresponding raw surfaces is merely the matter of a mattress suture. The suture material which we use is silk. The suture should be armed with two fine eye needles with a full curve. The first needle with suture is passed from the under or conjunctival surface of the lower lid and out through the excised area of skin then through the raw or undersurface of the upper lid to the outside. This procedure is repeated with the needle at the other end of the suture at a distance of about 4 mm. from the point of entry of the first needle. The procedure is illustrated in the accompanying diagram.



It is wise to tie the two ends of the mattress stitch over a small ball of cotton so as to avoid cutting into the skin. A short continuous stitch may be taken to approximate the margins of the lids. This is however not necessary if there is proper approximation with the mattress stitch. The eye or eyes should be kept bandaged for about ten days. The mattress stitch is usually removed on the twelfth day. The eye may be washed with weak anti-septic solutions after the fourth or fifth day. Of course the less meddling there is after the operation the less danger there is of non-union of the two surfaces. When dressings are changed it is important to avoid any pulling or tugging. Before removing the eye pad it should be thoroughly soaked with warm sterile boric lotion.

The cosmetic result of the operation is better if a bilateral canthorrhaphy is done. It is however not always necessary to do both sides.

The second method advocated by Kumbo adopts the same principle except that a triangular piece of mucous membrane is removed from each lid and with it the lash-bearing areas and the raw surfaces approximated as follows:

The stitches are inserted in such a way that four small pieces of rubber are brought in apposition to the stitches so that slight pressure is maintained. A piece of rubber about 2 mm. diameter is threaded on a needle with the suture stitches the needle is passed through the lower lid from without in and through the upper lid from within out leaving the rubber against the lower lid. When the needle is passed through the upper lid another similar piece of rubber is threaded. The needle then is taken through the upper lid as a mattress suture but before it is passed for a second time through the raw surface this time from without in a third piece of rubber is threaded in a similar way and then the needle passes through the upper lid and then through the lower this time from within out and finally the fourth piece of rubber is fixed in the same way the suture is firmly but not tightly tied. No bandage is needed but the eye should be washed daily and inspected to see that the rubber is not pressing on the lid. If this is so it is easy to remove one or more pieces of rubber and at the end of the fourteen days

the stitches are taken out. If lagophthalmos has been present for a considerable time not infrequently as a result of the irritation a small corneal ulcer develops. This may remain unnoticed because of the absence of pain and should be carefully looked for in all cases of paralysis due to neural leprosy. Sometimes the ulcer is not noticed until the inflammation has subsided and then a small ulcer is noticed on the cornea. Treatment should be instituted at once lest complications arise but these are not so common as ulcers resulting from acute iritis due to the *M. leprae*. The only method advised is cauterisation of the ulcer and this should be done as follows.

Place the patient in a good light and on a table. Cocainise the eye well then insert a speculum in order to control the eye one drop of fluorescin having been previously placed in the eye and the conjunctiva and especially the fornices well dried. Then dip a pointed match stick in carbolic and touch the edge of the ulcer all round and in the centre. Wherever the carbolic touches an opacity forms care must therefore be taken but over precaution will result in the process having to be repeated therefore be careful but do not be over cautious. Wash the eye out with saline and do not bandage. After treatment consists of atropine drops and saline washes until the ulcer is healed and all inflammation has subsided.

Treatment of Lepromatous Lesions of the Eye

In Chapter VI it has been pointed out that any or all of the elements of the eye may be involved in the lepromatous process either by direct spread from the skin or through the nasolachrymal duct or by the blood stream. In the description of the treatment of the eye in lepromatous leprosy we shall consider the involvement of each structure in turn.

A THE SCLERA

(a) *Diffuse Episcleritis*

It has been pointed out that this can be acute or chronic in the former the conjunctiva is always involved and there is therefore an associated conjunctivitis. Treatment consists of irrigating the eye with saline and applying the following ointment.

Sulphapyridine	grs. V (0.55 grm.)
Vaseline ad	$\frac{3}{4}$ 0.21 (30.0 grm.)

The ointment is continued for four days after which the eye is washed out with saline and a 25 per cent adrenalin solution instilled every four hours. If there is pain and lachrymation then dark glasses or eye shades are advised and a line of treatment adopted which is similar to that for iritis.

(b) *Chronic Diffuse Episcleritis*

The eye is washed out with saline and it will be sometimes found that trypan blue injected sub conjunctivally twice a day may be of value. The method is described under sub acute iritis. The injection is repeated when the discolouration is almost gone this takes about three weeks. If the cornea is invaded by the lepromatous granulomatous tissue extending from the sclera then it is advisable to make an incision completely around the perimeter of the eye this is best done with a pair of straight iris scissors and the incision is taken down to Descemet's membrane. There is some

evidence that leproma will not invade the scar tissue thus produced. The operation must however be done early and is indicated whenever the whole circumference of the cornea sclerotic junction is involved. A diffuse scleritis will occasionally clear up as the leprosy improves and is a condition which of itself is not serious. It becomes serious when it extends and the process invades the cornea or when complications—e.g. iritis—set in.

In circumscribed lepromatous lesions of the sclera a nodule gradually develops often situated at the limbus. This should be excised under local anaesthesia and the base well cauterised. The operation is very satisfactory when the nodule is hard and fibrous but when soft the nodule is not so easily removed the edges not so defined and there is a greater tendency for the process to become diffuse.

B IRITIS

This is divided into

(a) Acute iritis

(b) Sub acute iritis

(c) Chronic iritis

(a) Treatment of Acute Iritis

These cases should be treated along the same lines as acute lepra reaction as far as the general treatment is concerned. That is they should be put to bed given calomel at night and mag sulph in the morning. Then anodynes such as indicated in the chapter on Reaction in Leprosy should be prescribed. For pain in the eye compral appears to be most effective followed by a bromide mixture at night. Mercurials to reduce pain should include hot compresses of saturated mag sulph occasionally cold compresses act better. Leeches sometimes are of great value and should be applied to the temple. The leech is placed in a test tube the temple area scarified in order to draw blood or a drop of milk placed on the temple the test tube is placed against the area and withdrawn when the leech has taken a firm hold. The most important measure in the treatment of iritis is to endeavour to produce maximum dilatation of the pupil in the minimum time remembering that it is not easy to dilate a pupil when the iris is inflamed. Atropine 1 per cent should be instilled every three hours or if that is not sufficient atropine combined with scopolamine. Do not forget to exercise firm pressure on the nasolacrimal duct to prevent excessive absorption of atropine and unpleasant symptoms arising such as dryness of mucous membranes and giddiness. Once full dilatation of the pupil has been achieved the drops can be replaced by atropine ointment (1 per cent). The eyes should be cleansed with saline before drops are put in. Dark glasses or an eye shade should always be used for there is usually intense photophobia but bandaging the eyes is best avoided. Treatment should continue as long as there is any photophobia and even after the acute attack the pupil must be watched and dilated periodically or anterior and posterior synechiae will ultimately develop. If a mixture is thought necessary then atropine can be prescribed. In severe cases protein shock may be tried but caution must be exercised and therefore preparations such as aolin or lactolan should be given. Two c.c. intradermally every third day is considered better than 5 c.c. intramuscularly once a week. Where aolin or lactolan is not available then injection of sterile milk intramuscularly 2 c.c. increasing by 0.5 c.c. every week.

till 5 c c is reached may be tried Where cow s milk is poor in quality buffalo s milk should be used

Potassium iodide should under no circumstances be given

(b) *Treatment of Sub acute and Chronic Iritis*

This may be the result of a badly treated acute attack or the affection of the eye may pursue a slow and progressive course especially in chronic iritis hence the danger in such cases for persons who are illiterate and who do not usually read is that they may not realise that their vision is being affected until the damage is done Even so it is remarkable how much vision can be maintained in spite of extensive damage

In this condition photophobia and lachrymation are not marked symptoms especially if the iritis is chronic The tolerance of the eye to leprosy infection is some times remarkable Whether the iritis is sub acute or chronic the general principles are the same endeavour to dilate the pupil and maintain the maximum dilatation possible

Atropine (1 per cent) drops are instilled three times a day for two or three days and this is followed by atropine ointment (1 per cent) for a week If the pupil is not dilated by the end of the week scopolamine and atropine may be tried When the pupil does not dilate satisfactorily even after this the following procedure is suggested

Cocaine (3 per cent) solution is instilled wait four minutes and then adrenalin 1 : 1 000 is given wait another four minutes and then use atropine drops (1 per cent) and after half an hour repeat the procedure This may be done once a day followed on two or more occasions by atropine ointment or atropine and scopolamine ointment If the pupil still does not dilate satisfactorily the following subconjunctival injection may be given

R Atropine sulphate	gr	$\frac{1}{2}$	(0.016 gm)
Cocaine	gr	$\frac{1}{2}$	(0.03 gm)
Liq adrenalin (1 : 1 000)	ml	ΔLV	(3.00 ml)
Distilled water	5	14	(6.00 ml)
<i>Dose 3-5 ml</i>			

Atropine cocaine solution should be made up and sterilised first after which liq adrenalin is added

After the injection wait two hours and follow up with double atropine treatment as outlined above every day for a week If the pupil does not dilate after this then there is no hope of improvement without an iridectomy but continue atropine until such time as the eye is quiet enough for an operation

In sub acute iritis where there is more circumcorneal inflammation and greater lachrymation and pain before any subconjunctival cocaine adrenalin atropine solution is injected trypan blue should be tried One injection of trypan blue sub conjunctivally sometimes has a dramatic effect in relieving pain and lachrymation The eye is cocainised and the patient placed on the table in a good light The eye is controlled by an assistant and 3-5 mins of a 0.1 per cent solution of trypan blue injected subconjunctivally This is followed by one of the above methods used to produce dilatation of the pupil

Sub acute iritis in contrast to chronic iritis usually shows circumcorneal injection some photophobia and pain whereas in chronic iritis the main sign is a bound down and fixed pupil

If there is associated conjunctivitis then the eye is washed out with saline or boric lotion. In an intractable sub acute or chronic iritis a course of gold injections may be found useful remembering to give small quantities. The dosage has already been indicated. It is a good practice to use Hg_2O or flav at night and dionine and boric as indicated below may be found useful in sub acute iritis accompanied by conjunctivitis and keratitis.

C. KERATITIS

This may be of two varieties superficial and deep. The affection may be confined to a few maculae or there may be more extensive damage and opacities form which are gradually progressive and ultimately involve the whole cornea. This condition is serious and ultimately leads to total blindness. Various methods have been employed to endeavour to clear it up but its treatment is not very satisfactory and if the infection can be stopped from spreading then something has been achieved. It must be remembered that keratitis is always associated with iritis and therefore every effort must be made to dilate the pupil and maintain dilatation at the maximum. The following treatment may be tried in this condition.

Dionine and Boric

This is particularly useful in superficial keratitis associated with inflammation of the conjunctiva and sclera. Two per cent solution of dionine is used increasing gradually to 4 per cent. The strength is increased as soon as the eye ceases to lachrymate with the weaker solutions. Ultimately dionine and boric powder equal parts can be used placing a small quantity of the powder in the fornix and repeating the application once a day. Persistent treatment is useful when the opacities are small and in sub acute and chronic inflammatory conditions of the cornea and sclera.

If there are no inflammatory conditions and where the keratitis is superficial sub conjunctival injection of normal saline may help to clear the opacities. The dose is 0.2 cc increasing each week until 1 cc is reached. In deep keratitis care must be exercised for sometimes subconjunctival injections are found to be harmful.

Where patients are undergoing out patient treatment or if treatment needs to be continued over a long period then 1 per cent quinine bisulphate ointment may prove of some value. More than six subconjunctival injections of saline are not advised. When the course is over then quinine bisulphate ointment or dionine and boric powder should be continued daily.

In all forms of keratitis it should be assumed that there is an associated iritis and therefore the pupil should be dilated and atropine administered as indicated under sub acute iritis. When the eye is quiet and the opacity obstructs vision then iridectomy sometimes is of help but great care must be exercised in choosing cases for operation.

Operative Procedures

A word of warning is necessary in connection with operations on eyes which have become invaded by the M. leprae. Unless the operation is to save sight ophthalmologists should exercise the greatest caution before deciding to interfere surgically for three reasons.

(1) It is almost impossible to estimate when the eye is quiet and haemorrhage into the anterior chamber is a common complication in all operations involving the iris.

(ii) The iris is very friable and hence iridectomy is difficult to perform

(iii) An incision into the eye with any keratitis in leprosy or if the cornea is anaesthetic is so often followed by an extension of the opacity and ultimately loss of sight that many consider the risk too great unless the patient has already lost his sight and the operation is performed as a last resort

When all the elements of the eye are involved and the eye is useless unsightly and painful then enucleation should be performed

Glaucoma in Leprosy

Usually in leprosy the eye is soft and seldom does one find the tension raised and therefore there is little fear in producing secondary glaucoma under the intensive atropine medication recommended. The probable reason for this is because leprosy involves the ciliary body early and destroys or inhibits its function. In acute and sub acute iritis occasionally there is experienced a rise of tension then atropine must be stopped and active measures taken to reduce the tension hence when there is acute or sub acute iritis and treatment is started for the first time then homatropine should be tried first. Sometimes one finds in acute or sub acute iritis an eye may become sensitive to atropine. If so atropine should be stopped and when the pupil has recovered careful medication with homatropine should be started. It may be found that after a while the eye may be able to stand the full treatment laid down for this condition. No hard and fast rules can be laid down each case must be treated as individual experience dictates.

The fundamental principles in treatment of eye lesions are

- (i) Cleanse the eye by frequent douching
- (ii) Relieve pain and protect by eye shade or dark glasses
- (iii) Dilate the pupil by every possible means
- (iv) Keep every case under careful observation and if the iris is damaged dilate periodically, say once a month to prevent further adhesions

When colour vision is affected and there is an associated keratitis the outlook is extremely grave. It must be said however that a great deal of palliative treatment can be done and if blindness is not stayed off in many cases at least the day when the eye becomes completely sightless painful and useless may be considerably delayed. Patient persistent intelligent treatment brings in handsome dividends for it maintains the vision of the patient possibly for many years and earns his gratitude.

II TREATMENT OF LESIONS OF THE NOSE, MOUTH AND THROAT

(a) Lesions of the Nose

It is extremely important periodically to examine the nose in all cases of leprosy especially in lepromatous cases. Neural leprosy except in the tuberculoid case gives rise to no nasal symptoms. In tuberculoid leprosy however much discomfort may arise as a result of swelling and oedema of the mucous membrane causing blockage. Treatment is undertaken to reduce the inflammation and thereby establish free drainage. Where there is much swelling of the mucous membrane and the damming back of the

secretions then a warm alkaline douche should be prescribed. Following this the swollen mucous membrane should be treated by a solution of ephedrine (3 per cent) to which can be added 1 c.c. of 1:1000 solution of adrenalin to every ounce of the mixture. This mixture is best placed in an atomiser. Benzedrine and other vasoconstrictors may also be found useful when available. When the acute reaction condition subsides then all nasal symptoms also are alleviated but these measures will help relieve the temporary discomfort which arises through blockage of the nasal passage in the acute reaction of tuberculous leprosy.

In lepromatous leprosy in the early stages where there is no gross pathology of the nose it is probably better not to treat the nasal condition at all for as the skin becomes less positive so the *M. leprae* tend to disappear from the nose. If however the nasal smears are consistently positive and the skin tests show considerable improvement then the following nose drops may be prescribed for they may hasten the disappearance of bacilli from the nasal mucous membrane.

R	Creosote	5	II	(8.0 ml)
	Camphor	grs	XXX	(8.0 grm)
	Hydnocarpus oil		I	(30.0 ml)
	Olive oil	3	II	(60.0 ml)

If the mucous membrane is swollen then an alkaline douche followed by an ephedrine and adrenalin spray will help to reduce congestion and the above nose drops should be inserted after spraying the nose.

When crusts form and there is severe blockage of the nose the following procedure is advocated.

- Liquid paraffin is inserted the night previously to soften the crusts.
- Warm alkaline douche (soda bicarb. drim 1 to 1 pint) in the morning.
- The nasal passages are thoroughly dried.
- The camphor creosote hydnocarpus oil drops are inserted in each nostril.

If there is associated oedema and swelling of the mucous membranes between procedure (c) and (d) the nasal passages are sprayed with the adrenalin ephedrine solution. There may also be epistaxis when this occurs the nose is cleansed gently with warm saline or soda bicarb. solution and plugged with cotton wool soaked in adrenalin. No vigorous douching should be attempted until all signs of bleeding have ceased. If there are old signs of haemorrhage and ulceration then after the nasal douche the passage should be sprayed with 1 per cent glycerine and tannic acid or the following drops inserted.

R	Glycerine	II parts
	Iodine	1 part

When there is ulceration and the nose is cleansed and dried as above some advocate the touching of the ulcers with chromic acid (5 per cent) or with a chromic acid beard. Muir (1943) has recommended anaesthetising the nose and applying 10 per cent trichloracetic acid with a fine brush repeating after two weeks. We have had no experience of such treatment.

Occasionally in both the acute condition of the mucous membrane resulting from

The strength 1:3 per cent ephedrine with 1 c.c. of 1:1000 solution of adrenalin added to every ounce of ephedrine solution.

a tuberculoid reaction or in lepromatous leprosy the whole process may be complicated by a concomitant sinusitis which may be the result of the secretions and secondary infection of the sinuses. General symptoms of sinusitis are noticeable viz pain discharge and possibly oedema. The postural treatment according to the Proetz Parkinson technique should then be tried.

The patient lies on a bed or table on his side with a pillow under the shoulder and his head bent over it. He is told to breathe through his mouth while a few drops of the following solution previously warmed are instilled into each nostril.

1. Ephedrine hydrochlor	grs IV	(0.25 gm)
Sodium chloride	grs IV	(0.25 gm)
Aquam ad	3 I	(30.0 ml)

The patient continues to breathe through the mouth without swallowing for 2-3 minutes during which time the nasal mucous membrane shrinks and the ostia become more patent. After this period the patient's nostrils are closed with the balls of his thumbs and he is instructed to close his mouth and attempt to inhale vigorously. This manoeuvre results in the solution entering the sinuses after which the patient is turned downwards and the excess solution drains from the nose.

Plastic operations for the repair of nasal deformity have not been attempted on a large scale firstly because all such operations depend on the quiescence of the disease and secondly surgeons have not devoted much attention to the subject. We feel however that here is a profitable field for research.

(b) Lesions of the Mouth and Pharynx

As described in an earlier chapter these lesions are painful and distressing. In all cases vitamin B complex especially riboflavin should be prescribed. This can be given in the form of yeast (one ounce per diem) or one or other of the proprietary brands of tablets may be prescribed or if the patient is able to take it a whole wheat diet sprouting gram or other high vitamin content food. Such a line of treatment is advocated because it is difficult to differentiate a glossitis from leprosy involvement and a pure aribinoflavinosis. Apart from adding food rich in riboflavin the mouth should be kept clean and swabbed periodically with glycerine and borax or glycerine and tannic acid (24 per cent) if the mucous membrane is not too sensitive. Instead of swabbing the mouth and nasopharynx this may be sprayed with glycerine and tannic acid especially when there is evidence of leprosy granulation tissue. Infections of the teeth and pyorrhoea aggravate such conditions and therefore oral sepsis must be dealt with and dental care and hygiene emphasised. It is reported that promin or diasone helps considerably in this painful and distressing sequelae of oral leprosy.

(c) Lesions of the Throat

As pointed out in an earlier chapter throat lesions in leprosy are not only serious but are dangerous or potentially dangerous to life. Where there is much swelling and oedema an ephedrine spray should be tried. The nozzle of the de Vulliam spray is turned down and the patient asked to inhale. As he inhales the back of the throat is sprayed with the lotion. An alternative remedy especially when there is ulceration is glycerine and tannic acid (24 per cent). If there is hoarseness and not much evidence of inflammation hydnocarpus oil (pure) should be instilled into the larynx by means of

a laryngeal syringe. As modern methods of direct laryngoscopy are perfected a wide field of possibilities we hope will be found in the treatment of leprosy of the throat which at present is so distressing and for which so little can be done.

The fear of every physician dealing with advanced leprosy of the throat especially of the ulcerative variety is oedema of the glottis which may necessitate an immediate operation with a high mortality. Unless all other measures fail to relieve the obstruction tracheotomy should not be attempted in the acute stage. As a measure of temporary relief the patient should be given an injection of adrenalin and if much secretion adrenalin and atropine and inhalations of tr. benzoin. co. and a steam kettle will be found useful. Under no account should morphia be given to relieve distress as this depresses respiration and frequently gives the patient a feeling of impending death. When the acute condition subsides a low tracheotomy is attempted but if the oedema of the glottis is not relieved immediately then an emergency tracheotomy must be done. This is a simple operation in the quiescent stage but is so often happens patients wait till the condition becomes acute then the operation is fraught with difficulties.

Tracheotomy

This is adequately described in textbooks of surgery. One or two points however might be emphasized. The patient should be prepared overnight by the administration of luminal grs. 11. Just before the operation $\frac{1}{16}$ atropine but no morphia is given. The operation should not be unduly hurried. The head is placed over a block under the nape of the neck or on pillows and the trachea stretched sufficiently to expose the site of operation but care must be taken not to produce acute distress. It is better to have less convenience operating than a struggling frightened patient. The first steps are taken quietly but not too rapidly and be sure of the completion of every step before going on to the next. The operation is done under a novocaine block. It is better to block a rectangular area leaving the line of incision free from novocaine solution for if much fluid is injected it obscures the field. An incision is made in the midline and care is taken that an assistant holds the head straight and lightly stretched. A dissection mostly blunt is made down to the trachea catching any bleeding points but not being excessively worried over venous oozing. This will stop as soon as the trachea is opened. It is very important to be certain that the trachea is exposed. Beginners tend to go too high and miss the ring of the trachea. When the trachea is exposed steady it with one finger and plunge the knife between the rings with a sharp jab and twist the blade so as to maintain the opening patent then insert the tracheal dilators. Once the care is in allow the patient to cough up collections of mucous and pus. The operator must remember to keep his mouth and eyes well out of the way for the explosive force of the cough when air enters the trachea is considerable. When the air passages are clear carefully insert as large a tracheotomy tube as possible. The rest of the operation can be completed more leisurely. The tube is tied at the back of the neck by tapes. bleeding points dealt with a few interrupted silkworm sutures inserted the operation area painted with acriflavine or dusted with sulphonamide powder and the patient returned to bed. The inner tube is kept in for twenty four hours unless blockage occurs then it is carefully removed and the blockage relieved. The outer tube remains for three months (see below for hygienic technique during this period).

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The fear of every physician dealing with advanced leprosy of the throat especially of the ulcerative variety is oedema of the glottis which may necessitate an immediate operation with a high mortality. Unless all other measures fail to relieve the obstruction tracheotomy should not be attempted in the acute stage. As a measure of temporary relief the patient should be given an injection of adrenalin and if much secretion adrenalin and atropine and inhalations of tr benzoin and a steam kettle will be found useful. Under no account should morphia be given to relieve distress as this depresses respiration and frequently gives the patient a feeling of impending death. When the acute condition subsides a low tracheotomy is attempted but if the oedema of the glottis is not relieved immediately then an emergency tracheotomy must be done. This is a simple operation in the quiescent stage but as so often happens patients wait till the condition becomes acute then the operation is fraught with difficulties.

Tracheotomy

This is adequately described in textbooks of surgery. One or two points however might be emphasised. The patient should be prepared overnight by the administration of luminal grs ii. Just before the operation 100 atropine but no morphia is given. The operation should not be unduly hurried. The head is placed over a block under the nape of the neck or on pillows and the trachea stretched sufficiently to expose the site of operation but care must be taken not to produce acute distress. It is better to have less convenience operating than a struggling, frightened patient. The first steps are taken quickly but not too rapidly and be sure of the completion of every step before going on to the next. The operation is done under a novocaine block. It is better to block a rectangular area leaving the line of incision free from novocaine solution for if much fluid is injected it obscures the field. An incision is made in the midline and care is taken that an assistant holds the head straight and slightly stretched. A dissection mostly blunt is made down to the trachea catching any bleeding points but not being excessively worried over venous oozing. This will stop as soon as the trachea is opened. It is very important to be certain that the trachea is exposed. Beginners tend to go too high and miss the ring of the trachea. When the trachea is exposed steady it with one finger and plunge the knife between the rings with a sharp jab and twist the blade so as to maintain the opening patent then insert the tracheal dilator. Once the care is allowed the patient to cough up collection of mucus and pus. The operator must remember to keep his mouth and eyes well out of the way for the explosive force of the cough when air enters the trachea is considerable. When the air passages are clear carefully insert as large a tracheotomy tube as possible. The rest of the operation can be completed more leisurely. The tube is tied at the back of the neck by tapes. bleeding points dealt with a few interrupted silkworm sutures inserted. the operation area painted with acriflavine or dusted with sulphonamide powder and the patient returned to bed. The inner tube is kept in for twenty-four hours unless blockage occurs then it is carefully removed and the blockage relieved. The outer tube remains for three months (see below for hygienic technique during this period).

Complications

When the operation is done as an emergency the mortality is high and death may occur on the table through shock. Occasionally the patient does not breathe when the trachea is opened and the tube is inserted. Artificial respiration should be started a catheter passed and mucous sucked out of the trachea. This may result in the removal of the blockage.

Complications arising within twenty four hours to seventy two hours are

(a) Surgical emphysema (b) Haemorrhage (c) Aspiration pneumonia

(a) *Surgical Emphysema* This is usually due to the tube either not being inserted properly or being displaced by movement. The patient should be taken back to the theatre and the tube be reinserted. Do not attempt to reinsert the tube when the patient is in bed for difficulties may arise which can only adequately be dealt with in the theatre.

(b) *Haemorrhage* This is an unusual complication but the patient must be returned to the theatre and the bleeding point ligatured.

(c) *Aspiration Pneumonia* This fortunately is a rare complication but extremely serious. Sulphadiazine or penicillin should be given in adequate dosages.

Delayed Complications

If the patient survives a week he is likely to recover. Either due to extension of the ulceration or constant irritation of dust particles of mucous and crusts are liable to form. These can be prevented by careful technique in cleaning the inner tube. For this purpose the patient is taught to remove the inner tube which is washed with soda bicarb then placed in Dettol after which it is rinsed in sterile water dried and well lubricated with hydnocarpus oil. The oil will not only make the insertion of the tube easier but a little trickle finds its way down the trachea and helps to soften the crusts which are liable to form. After three months if no complications have arisen necessitating the removal of the outer tube the patient is taken on the table and the outer tube removed and the wound inspected. After this when cleaning the tubes can both be daily removed as the tracheotomy opening is now permanent.

While the operation of tracheotomy is a drastic one if successful it gives so much relief to the patient and makes the remaining days more comfortable that one should not hesitate to perform it. As soon as it is decided that laryngeal leprosy has advanced so far that there is no alternative to tracheotomy then an operation should be undertaken without delay.

TREATMENT OF TROPHIC ULCERS

Trophic conditions in leprosy result from injury to nerves and occur for two reasons

(a) Injuries which in the normal skin would not give rise to trouble in anaesthetic skin are not noticed and hence they become extensive and serious because the defensive reflex of pain is lost. Hence pressure blisters, burns both due to heat and idiopathic burns must all be considered under this head.

(b) Lesions resulting from the disturbance of the neuro trophic functions of a limb.

Both these factors usually operate simultaneously. For instance pressure may give rise to a blister on a pressure point such as the head of a metatarsal bone but owing to the trophic condition of the skin a perforated ulcer arises which quickly affects the bone.

The first sign although not constant of trophic disturbance in a limb is rarefaction of bones and this may occur without any evidence of ulceration. As a result of such trophic disturbance there seems to be a local loss of calcium resulting in a further degree of rarefaction and then absorption of bone. This absorption takes place whether there is actual ulceration or not. It is interesting to note that digits do not drop off. They are absorbed and even when all the phalanges or the carpal or tarsal bones become absorbed there is still little evidence of the five finger nails at the distal end of the stump.

Actual necrosis and sequestra formation with compensatory callus formation is never seen apart from sepsis. The sequel is anaesthesia injury, sepsis, necrosis with or without sequestra. As will be pointed out under operative procedures sepsis is less of a menace in leprosy than in the healthy individual and contrary to the generally accepted opinion anaesthetic areas frequently heal and granulate better than normal skin. This has been explained by the fact that changes in the walls of the veins are more frequent than in those of the arteries in leprosy and hence an almost permanent state of Bier's passive congestion may be present in the limbs. Not only is sepsis less common but healing is sometimes more efficient. It is impossible in this chapter to deal in detail with X ray changes in bones. Readers who are interested are referred to the excellent article in *Radiology* by Fadget and Maimot (1944).

If then it is the case that no necrosis of bone will result unless there is previous injury it is absolutely essential to endeavour to prevent injury and damage to soft tissues which ultimately leads to trophic ulceration, extension of bone and necrosis of the phalanges, metatarsal or tarsal bones. It cannot be too strongly stressed that patients who have anaesthesia must exercise the greatest care. For instance they should be warned not to undertake any work which involves the handling of hot utensils e.g. cooking vessels. When they drink hot fluids they should be instructed to take care that they do not hold hot brass vessels or allow the hot liquid to spill over their hands. In this connection nurses or attenders who care for patients with anaesthetic leprosy should be reminded that what is pleasantly hot for a normal individual is too hot for a person with anaesthesia. Warnings should also be issued to patients against

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knocking their feet or hands for slight trauma will raise a blister which if over a metatarsal bone may result in a trophic ulcer and extension to bone with resultant necrosis. Massage exercise avoidance of any injury and cleanliness are the secrets of prevention of damage in neural anaesthetic leprosy. If in addition it is remembered that crevices between the toes should be kept dry and every possible precaution taken to prevent excessive perspiration then even when a foot is severely damaged it is remarkable how much function can be retained.

With this introduction we pass on to the more detailed consideration of trophic ulcers. Trophic ulcers in general can be divided into those ulcers which do not obviously affect bone and those which have extended down to bone which can usually be felt



FIG 1 2.—Extensive ulceration of foot with much sloughing



FIG 1 3.—Ulcer healed as a result of conservative measures

at the base of the ulcer. Further under trophic conditions must be included burns and injuries which in normal individuals would cause little or no damage but result in extensive ulceration in patients suffering from leprosy. In the treatment of all ulcers whether they affect the bone or not whether they are the result of slight or marked trauma the fundamental principles are to cleanse the ulcer and endeavour to encourage the formation of healthy granulation tissue and then to protect the granulation and thus aid healing. The remedy which has been found of most value for cleansing ulcers and encouraging the formation of healthy granulation tissue is the well known eusol lotion. It is our experience however that for eusol to exercise its maximum benefit the dressing should be changed two or three times a day. Further it should be borne in mind that the dressing should be applied as a pad and that the gauze should not be drenched in the lotion. It is recommended that pieces of gauze should be soaked in eusol and wrung out so that excessive fluid does not run down and soak all the surrounding tissues for if it does an unhealthy condition of the skin results and thus forms an excellent nidus in which septic organisms can flourish. If

eusol is not available or if the patient cannot get frequent change of dressings then the remedy which is found most cleansing is eucalyptus oil and iodoform. This remedy as far as is known was first used some years ago by Dr Lang then of Chandkuri.

The formula is as follows

R Iodoform	grs $\frac{1}{5}$	(0.6 grm)
Eucalyptus oil	I	(30.0 ml)

When the formation of granulation tissue has commenced the dressing sometimes sticks owing to the tendency of the eucalyptus oil to evaporate. If this is troublesome equal parts of castor oil or olive oil is added.

Workers in institutions have often to admit persons with foul smelling ulcers associated with oedema threatening or actual cellulitis and much pus formation. The pus is frequently found to be oozing from multiple sinuses. Our usual practice is to soak the limb in warm saline or in hypertonic saline. Potassium permanganate may be used to reduce the foul odour but should be changed as soon as the lotion turns brown. Where there is much oedema

and sloughing saturated magnesium sulphate baths are used. There are one or two points to be borne in mind when prescribing such a treatment. Firstly, remember to test the temperature of the water before immersing a limb for the patient cannot appreciate heat and secondly after soaking which should not be for more than ten minutes gently remove all sloughs. The sloughs usually separate easily but may take several days to come away completely. After removing the



FIG 14—Ray photograph of foot showing ulcers and sloughs before and after treatment.

sloughs dry the limb thoroughly and spirit it well so that excessive moisture is not left to encourage bogging of the tissues. Finally raise the leg and apply strips of gauze which have been soaked in eusol lotion and wrung out to remove excessive fluid. Bandage and immobilise the limb by splinting. Sloughs can sometimes be caused to separate more quickly if the ulcer is dressed with sterile magnesium sulphate powder. Dressings should be changed three or four times a day if necessary. Until the ulcers are clean and the oedema subsides the foot of the bed should be raised. Where pus is extending and cellulitis is threatened incisions should be made. We usually find multiple small incisions preferable to larger ones. If there are any sinuses these should be douched with eusol or saturated mag. sulph or syringed out with Dettol (1.0 solution) as advocated by Ryrre (1936). It is usually found that in trophic conditions associated with oedema and cellulitis that once the septic condition is controlled the ulcers begin to granulate freely and not infrequently excessively. When the temperature remains high indicating septic absorption then the sulphonamide products are indicated. It should be remembered that sulphonamide preparations and of these sulphathiazole or sulphadiazine are the most useful should not be given unless or until there is free drainage or pus. Frequent differential counts should be done particular attention being given to the total white count and the relative number of the polymorphonuclear leucocytes.



FIG 15—Trophic ulcer head of first metatarsal bone but not extending to joint and bone not exposed

excessive oozing we usually continue either eucalyptus oil and iodoform ointment is applied

R Acid borie
Oil eucalyptus
Bismuth subn
Zinc oxide
Castor oil

grs	VV	(10 grm)
℥	V	(10 ml)
grs	LX	(40 grms)
grs	LXXX	(120 grms)
℥	I	(300 ml)

If granulations are excessive painting with 2½ per cent aqueous solution of silver nitrate or touching the granulations with a silver nitrate stick or with copper sulphate is sometimes effective

There are then the general measures adopted in treating trophic ulcers with no marked bone involvement. The only further point to stress is that whereas we do not hesitate to sacrifice a toe we refrain from sacrificing a finger or even part of a finger as long as possible and remove the phalanges only when it is absolutely unavoidable for a deformed hand or finger is usually better than none at all. It is remarkable how seldom one needs to undertake operative interference in the case of infection of the hand or fingers except local incision for purposes of more adequate drainage.

Lowe and Chatterjee (1937) recommended injecting trophic ulcers once a week with hydnocarpus oil. These workers as well as others have reported considerable success with this procedure. There are two methods of giving the injections into the ulcers and these may be called the superficial and the deep methods. In the former the needle is inserted into the layer just under the plantar fascia but the point does not penetrate into the deeper layers and multiple injections are given all round the ulcer not more than 1 cc being injected at one sitting. In the deep method on the

In foul smelling ulcers associated with necrotic bone we do not think it advisable to remove the bone or scrape it until the sepsis has been controlled. Operation if undertaken should only be confined to measures which help drainage. While sepsis is usually of a low grade occasionally a virulent streptococcal infection is encountered. This may result from fresh injury or from spreading of an infective process due to attempting extensive surgical procedures before the sepsis is controlled. In leprosy when this occurs the situation is always grave and the mortality high. All measures must be taken to localise the infection and with the availability of penicillin on a large scale the mortality from a virulent streptococcal infection should be reduced considerably.

When the sepsis has been controlled and a clean granulating surface produced and healing commenced then a protective dressing is applied. It should be remembered however that neither an oily dressing nor an ointment can be applied if there is much oozing. In the presence of

dressing with eucal until this has stopped. Then with equal parts of castor oil or the following



FIG 16—After preliminary anesthetic, ulcer injection with hydrosorptol oil. One week. Healing (see Fig. 15).



FIG 17—Injection continued. Completely healed at the end of six weeks.



FIG 18—Trophic ulcer at the base of index finger (occupational). This type of ulcer rarely seen on the finger but is similar to the foot and the leg ulceration treatment.



FIG 19—Ulcer healed on injection treatment a month later.

other hand the needle is inserted through the surrounding normal tissue penetrating deeply into the tissues of the foot and pointing towards the centre of the ulcer the injections are given diagonally the proximal and distal margins of the ulcer being injected one week and the inner and outer the next week Half c c to 1 c c of the medicine is injected at each puncture After the injection the ulcer is dressed with the same remedy which is used for the injection Hydrocarpus oil is the most convenient substance but cod liver oil is equally effective and the ulcers heal satisfactorily with either remedy The two main precautions necessary are never to inject a dirty ulcer and to cleanse the surrounding skin and base of the ulcer adequately for if the wound should become infected a considerable amount of sloughing may result



FIG. 130.—Ulcer over heads of first and second metatarsal bones suitable for injection treatment

All ulcers do not respond to injection treatment and indiscriminate injection of ulcers will only bring an excellent method into disrepute Generally speaking where there is much thickening of bone and especially if there is much pain associated with periostitis or if there is not an adequate covering of subcutaneous tissue over the head of a metatarsal bone injection treatment will fail The ulcers which heal most readily are the clean perforating ulcers with thickened skin and without excessive necrosis of bone or periostitis Injection treatment if precautions as to antisepsis are carefully taken will do no harm and is worth a trial before resorting to operative procedures While the deep method of injection in our opinion is the best with ulcers over the internal or external malleolus where there is no marked destruction of tissue the superficial method will be found to be more satisfactory but on no account should this be used if there is much sepsis oedema or inflammation of the surrounding tissues

From time to time strapping of ulcers has been advocated and Paul (1936) in Ceylon recommended a procedure similar to that used for varicose ulcers namely with the leg raised and the veins emptied overlapping pieces of adhesive strapping are firmly placed over the ulcer the strapping is left until it is ultimately separated by the gradual soaking of the exudate from the ulcerating surfaces The chief objection to this method is that the foul smell from the dressing is usually complained of by the patient and the nursing staff naturally dislike such a method If this method is resorted to we prefer filling the cavity with a 5 per cent sulphonamide paste bandaging or strapping and leaving it for a week The following is the prescription for sulphonamide paste

R	Sulphonamide	grms	5
	Adep lanac	grms	70
	Paraffin liqd	grms	25

The results of this method are encouraging, and where the ulcer is not excessively

dirty then this method gives dressing and promotes healing. For ulcers over the external or internal malleolus sulphuramide paste with splinting sometimes succeeds when all remedies have failed. The cost is not excessive when the saving of dressings is taken into consideration. Oberdoeffler and Collier (1939) have recommended the following ointment:

R Mercurochrome
Honey
Cod liver oil
Zinc oxide powder
Bismuth subn
Vaseline

II (60.0 gm)
VIII (40.0 ml)
VIII (240.0 ml)
IV (120.0 gm)
II (60.0 gm)
VII (300.0 gm)



FIG 131 — Ulcer healed after operation on trochanter



FIG 132 — External ulcer over first metatarsal head after operation on trochanter

The honey and mercurochrome ointment is very expensive and therefore in the Lady Willingdon Leprosy Sanatorium mercurochrome is replaced by eucalyptus oil and appears to be as satisfactory. The prescription is as follows:

R Oil eucalyptus
Honey
Cod liver oil
Zinc oxide powder
Bismuth subn
Vaseline

1 (15.0 ml)
II (60.0 ml)
II (60.0 ml)
I (30.0 gm)
1 (15.0 ml)
III (30.0 gm)

When an ulcer is dirty especially when the surrounding skin is affected and the surface not oozing application of ung. hydrarg. sometimes produces healing. When the surface of the ulcer is oozing and the edges boggy moist dressings are better avoided and

particularly if there are large painful ulcers over the lower part of the limb the following antiseptic dressing will be found useful

R Sulphonamide¹

Kaolin

Cod liver oil

Glycerine

grs	XXV	(1.66 grm)
grs	LX	(4.00 grm)
℥	ALA	(3.00 ml)
3	I	(30.00 ml)

The kaolin acts as an absorbent and the glycerine as an astringent. This cream will also be found useful in the so called chronic tropical ulcers.

When a good clean granulating surface is produced then acriflavine and paraffin



FIG. 133—Extensive ulceration and considerable sloughing over external malleolus



FIG. 134—Ulcer healed with sulphonamide paste and strapping

may be tried. It protects the newly formed granulations and produces healing. The method of preparation is as follows:

Dissolve 0.5 grms acriflavine in 25 c.c. of warm boiled distilled water. Sterilized wool fat 30 grms is put in a sterile mortar and the acriflavine solution is added in small quantities and finally liquid paraffin to 100.

For breaking down nodules of the legs and arms and when the granulating surfaces are clean the following modification of Unna's paste may be tried:

Unna's Paste

R Water
Gelatine (sheet)
Glycerine
Zinc oxide powder

℥	X	(0.6 ml)
℥	VI	(0.4 grm)
℥	X	(0.6 ml)
℥	VI	(0.4 grm)

¹ No particular sulphonamide preparation is suggested for recent research reveals fresh and more effective preparations and therefore the particular sulphonamide is left to the individual physician's choice.

In 10 oz of hot water (in a water bath) dissolve 6 oz of gelatine. Add 10 oz of glycerine while the fluid is hot (still in water bath). Add slowly 6 oz of zinc oxide finely powdered stirring slowly (still in water bath). Keep the paste warm until used.

Use when warm applying the mixture in a similar way as one applies a plaster bandage. The preparation hardens and can be left on from four days to a week or ten day according to the nature of the ulcers and the amount of exudate. This method became prohibitive during the war years.

Another method which also was advocated by Milroy Paul is putting the limb in plaster. When the ulcer is clean and when there is no discharge then by resting the leg in a plaster case and incorporating a stirrup iron into the plaster frequently results



FIG. 135—Ulcer suitable for injection treatment after metatarsal bone was removed.



FIG. 136—Scar left after removing the metatarsal bone and leaving the third toe.

in the healing of the ulcer. Before applying the plaster the limb must be raised and the engorged veins emptied. The patient should be given crutches. The plaster is kept on for a month or six weeks. Indications for removing the plaster are pain, discharge or a foul smell. It must be remembered that while superficial pain is lost the pain resulting from pressure either from the plaster bandage or from inflammatory products is not eliminated and therefore is a warning sign should anything go wrong.

When ulcers are healed and there is deformity resulting from the downward dislocation of the heads of the metatarsal bones then a metatarsal bar should be placed on the shoes if possible. This raises the arch of the foot and prevents excessive pressure on the heads of the bones and hence eliminates the greatest cause of ulceration namely pressure.

The operative procedure which is commonly resorted to apart from amputation of the phalanges is the removal of the metatarsal bones.

Indications

This operation should be done if the following conditions are present

(1) There is an ulcer leading down to the head of the metatarsal bone the bone being uncovered by granulation tissue

(2) Ulcers which have resisted all local measures and which involve not more than two metatarsal bones especially if the following signs are present

(a) A sinus leading to necrotic bone

(b) Pain on pressure over the metatarsal bone sometimes the bone is actually movable when grasped between the finger and thumb

(c) Characteristic blackening of the foot

(d) A ray evidence of irreparable damage to the bone

Another sign which is frequently present is a curious blackening of the skin over the necrotic metatarsals and along with other evidence this sign may be found useful

With regard to the removal of the metatarsal bones confirmation by X ray when ever possible is extremely important for it gives evidence of the extent of damage to other bones. A general principle is that one should be as conservative as possible so that the shape of the foot is maintained otherwise further damage will result as soon as the patient walks hence only when absolutely unavoidable should the first metatarsal be removed

If more than two metatarsal bones are involved then it is useless to remove them all conservative methods should be persevered with and if these fail further more drastic operative measures will have to be resorted to. The operation will now be described

The patient is prepared in the usual way the night before and gr ii of luminal (phenobarbitone) are given. If the patient is nervous $\frac{1}{16}$ gr of atropine and $\frac{1}{2}$ gr morphia may be given before the operation. The limb is devascularised by running it and emptying the veins by massage a tourniquet is then applied. When the tourniquet is applied the local anaesthetic is now injected. This is advocated for two reasons, first there is no fear of absorption of the local anaesthetic and secondly it remains longer in the tissues when a tourniquet is applied first and therefore is more efficient. The technique is as follows infiltrate the nerves which pass under the external and internal malleolus. Then a band of infiltration is produced right across the dorsum of the foot one inch above the proximal end of the metatarsal. Finally a deep injection is given between the metatarsal bones. If the second metatarsal is to be removed then the deep infiltration is placed between the first and second and the second and third. If the local infiltration anaesthesia is done properly the only pain from which the patient suffers is the dull pain of the tourniquet.

An incision is now made on the dorsum of the foot over the head of the metatarsal bone to be removed. The bone is exposed over the whole length and the periosteum incised. Confirmatory evidence of complete necrosis is seen in the ease with which the periosteum strips from the bone. If the periosteum does not strip easily then the bone is probably not completely necrotic. Always keep near to the bone and in this way the bone is freed from the surrounding tissues and both ends cleared. The distal joint is then entered and the bone separated from its phalanx. If the toe is not worth preserving this is removed at this stage but a more aesthetic result is achieved by leaving the toe if it is healthy. No trouble arises if the tendon is removed as the toe becomes fixed

down by firm fibrous tissue. When the distal joint is freed the proximal joint is then similarly dealt with. The metatarsal is now ready to be dissected out. Commence at the distal end and keeping near the bone gradually free it from adhesions and the underlying ligaments then the bone is seized with lion forceps and the final disarticulation is made. The edges of the wound are trimmed and the cavity well packed with gauze soaked in glycerine and iodoform. A tight bandage is then applied.

It is our practice not to remove the tourniquet until the wound has been well packed and a tight bandage applied and the patient put to bed with the foot of the bed raised. Secondary haemorrhage is rare. The cavity left after the removal of the metatarsal bone may be picked with 5 per cent sulphamizide paste leaving the dressing for a week. So far the results have been excellent even when a grossly infected bone has been removed. By using sulphamizide paste dressings are saved secondary haemorrhage is reduced to a minimum and healing is quicker. The dressing is changed once a week and the cavity repacked with sulphamizide paste. Frequently when using this paste there is on taking off the dressings a somewhat penetrating and objectionable odour but this does not necessarily indicate infection.

It is wise to keep in mind the following general principles

(a) As far as possible clean up the operation area of any acute sepsis

(b) Make a large enough incision in order to work properly and define the proximal joint before endeavouring to separate the bone

(c) Separate the bone from above and work downwards

(d) Try to avoid any injury to the articular surface of the cuneiform or cuboid bones

Recently Muir (1943) has advocated the plantar approach. We believe that such an approach is anatomically unsound for two reasons

(1) The incision involves the disturbance of the main blood supply to the foot

(2) After the removal of the bone the chief support is the plantar fascia and an incision through it may damage this and hence lead to further ulceration

In co-operation with Gass we have removed metatarsal bones from the plantar aspect and while we consider this approach not so satisfactory in cases where the head of the bone is so far dislocated that it appears in the wound the plantar approach exposes the bone better and the posterior ligament which is the most difficult to penetrate from the dorsal approach is more easily dealt with. When the plantar approach is used then the tourniquet must be loosened before the wound is stitched because as stated the main blood supply—the plantar arch—is in danger of being severed. This method is not recommended as a routine even though when successful healing appears to be quicker.



FIG. 137—X-ray photograph showing the involvement of the metatarsal bone of the foot. Not shown of the tarsal which has been removed. Operation of the foot would be an amputation through the joint between the tarsal and the metatarsal.

When more than two metatarsal bones are involved then more drastic operative procedures are necessary. A fundamental principle is to leave a weight bearing stump. So often deformity of the hand is associated with damage to the feet and if the patient has to use crutches one of two things is liable to occur

- (1) Damage to deformed and anaesthetic hands due to pressure from crutches.
- (2) Extra strain on the other limb which if anaesthetic and already damaged will break down.

The choice of operation should be decided if possible by X ray. If the smaller tarsal bones are involved and the tarsal metatarsal joint is intact then a Lisfranc operation should be recommended. If the small tarsal bones are affected and the joints between the navicular and cuneiform bones or the cuboid and cuneiforms are affected then a Chopart's amputation should be attempted. If the talus or os calcis is damaged then it is little use attempting either of these operations for sooner or later the stump will break down. Conservative measures such as rest and a plaster of paris cast should be adopted and operation postponed and if the ulcer extends or does not heal then an amputation should be performed in the lower third of the leg.

Cass advocates the Symes operation but our experience on the whole has been unfavourable largely because of the expense and difficulty of securing a good Symes boot. Even if this is obtained there is a great tendency to further absorption of bones through pressure resulting in a conical stump and permanent ulceration necessitating amputation at a higher level.

Lisfranc and Chopart's operations are now seldom performed in surgery therefore the main principles will be described. The patient is prepared in the usual way and the operation done preferably under spinal anaesthesia or if contra indicated under local novocaine block. An incision is made half an inch above the head of the fifth metatarsal bone. A short dorsal flap is reflected. The purpose of this is to expose the tarsal metatarsal joint. Theoretically the knife should be inserted into Lisfranc's joint behind the tubercle of the fifth metatarsal by strongly flexing the foot all the joints are then exposed but owing to the damage already done it is extremely difficult to deal with the joints in this simple manner. Therefore the heads of the metatarsal bones are all exposed the foot firmly flexed and the joint which is easiest of penetration penetrated first. Once one joint is penetrated then it is a simple matter to expose the rest of the joints remembering that the second metatarsal bone juts up a little because the corresponding cuneiform bone is shorter. When all the joints are penetrated and the ligaments severed take an amputation knife keeping the foot strongly flexed the knife pointing towards the toes and the blade near to the bones then fashion a large plantar flap. If the flap is too short and sometimes in any case the navicular bone juts out and is liable to make the stump uneven then this bone is better dissected out. Loosen the tourniquet deal with the bleeding points and stitch the large plantar flap to the skin of the dorsum of the foot. Dress with renflavine and return the patient to bed with a firm bandage.

Chopart's operation is done in exactly the same way but the incision passes through the articulation between the cuboids and os calcis the navicular and astragalus (talus).

Further points in this operation should be noted. While a clean operating surface is essential yet if the foot is badly deformed one need not wait for the complete healing of the trophic ulcers. These should be excised and the flap fashioned accordingly.

It is remarkable how often a stump heals in spite of trophic ulceration. If sepsis occurs it is usually mild. The central stitches should be removed leaving the ones at either side. These retain the stump in place and allow for healing by granulation tissue without distorting the stump. As far as leprosy is concerned the fear that the tendo achillis if left will produce a twisting of the stump is in our experience quite unfounded. In stitching the flap however care must be taken to stitch it as far as possible in the normal position of the foot for it is easy to twist the stump by not taking note of this and then further pressure points may result and in consequence the stump breaks down.

If a Lisfranc and Chopart's cannot be done due to extensive damage then a lower third amputation should be performed. When artificial limbs can easily be secured then the choice of the site of the amputation is influenced by this fact but when in the East good limbs are not available and when the patient so often insists on as little as possible being done then a lower third amputation is advised. After forty eight hours in the case of a Lisfranc or Chopart's operation the wounds should be inspected. If clean then dressings are continued with acriflavine. Should there be any signs of sepsis the necessary stitches are removed and the wound dressed with eusol twice daily. It is seldom that sepsis is alarming and it is amazing how well such flaps heal even in the event of the necessity of removing all but the lateral stitches. In fact in limbs affected by neural anæsthetic leprosy risks can be taken which in a normal limb would spell disaster and surgeons who are used to dealing with normal feet are liable to be over cautious.

It must be remembered that each physician has his own method of dealing with trophic ulcers and that one cannot have too many remedies at one's disposal because repeated changes have often to be made and it is the doctor who always has something up his sleeve who is most likely to be successful in the treatment of this distressing sequel of leprosy. We feel that if the general principles of cleansing protecting the granulating surface and ensuring adequate drainage and maintenance of the blood supply with partial or complete mobilisation of the affected limb are borne in mind then the majority of ulcers will heal. The more experience one has the more conservative does one's method become. Amputation is only justified when life is endangered or for a painful and deformed limb which will not yield to conservative methods.

CHAPTER XVIII

A TREATMENT OF SYPHILIS AND LEPROSY

B TREATMENT OF CERTAIN COMMON CONCOMITANT SKIN AFFECTIONS

A Treatment of Syphilis and Leprosy

It has too often been assumed that because a patient with leprosy has a positive Wassermann reaction he therefore has syphilis. In Chapter V evidence has been given which sets forth reasons for doubting this statement and strong support is furnished to the theory that a positive Wassermann or Kahn reaction may have no significance in leprosy and that physicians are not justified in giving anti syphilitic remedies in leprosy unless there are clinical signs of past or present syphilis or a history of infection.

It is admitted that in the above experiment the method of treating syphilis if it were present would not meet with the approval of syphilologists and therefore not until a standard course of treatment is undertaken can one categorically state that a positive Wassermann in leprosy is not due to a spirochetal infection. The most significant support however for the statement that advanced leprosy in itself causes a positive Wassermann is seen in the disappearance of a positive serological reaction when lepromatous leprosy becomes negative. Recently Faget and Hilary Ross (1944) have confirmed this opinion.

When dealing with large numbers of patients in a leprosy sanatorium or as out patients a physician will constantly come up against the problem of how to treat syphilis in the presence of leprosy. While it is known that treatment for syphilis may precipitate a reaction it is felt that owing to the seriousness of the disease one should not therefore give ineffective courses but should give the full course as laid down by specialists. If a reaction develops in the course of the treatment then anti syphilitic treatment should be suspended temporarily until the reaction subsides. While making this statement it cannot be too strongly emphasised that the physician must be certain that the patient is suffering from active syphilis for we do not believe that one is justified in giving a course of treatment which is expensive and may precipitate reaction unless there is unequivocal evidence of syphilis. To treat for syphilis on the assumption that a positive Wassermann reaction in leprosy is due to syphilis is neither beneficial to the patient nor to the reputation of the doctor.

The remedy recommended because it is known to be less toxic and more readily excreted by the kidney is mepharside. The dose should be calculated on the basis of 0.04 grm. of mepharside for every 100 lb. of body weight. This dose should be given intravenously every five days for twenty injections. Along with the course a ten week course of bismuth should be prescribed. The dosage should be calculated as 0.2 grm. of metallic bismuth and this is given intramuscularly once a week for ten weeks. At the close of the treatment one month's rest is advised and the Wassermann reaction taken. If the reaction is still positive the whole course is repeated. It is advised by some that four such courses should be given before one can assume that adequate treatment has been completed. I am aware that such a regime is drastic

and not free from danger of reaction and therefore it cannot be too strongly emphasised that clinical evidence of syphilis must be present before a physician should embark on its treatment in cases of leprosy. The combination of bismuth with arsenic helps it is believed to control every reaction.

The above regime should be carried out for all active cases of syphilis both in the primary or secondary stage. In cardiovascular or neuro-syphilis of under six months duration the above course should be preceded by six doses of bismuth prior to mapharside being given then the routine as advised is followed giving the remaining four bismuth injections making the total number of bismuth injections ten. It would probably always be well to follow this practice in the treatment of syphilis in leprosy.

In the treatment of children acetylarsen infantum is recommended and the following is the suggested dose recommended according to the weight of the infant.

Above 2 ½ lb	1 ½-2 c c
Between 15-20 lb	1-1 5 c c
Below 10 lb	0 25-0 5 c c

The injections are given twice a week intramuscularly and a total of twenty injections are given. This is combined with 0 5 c c of bismuth once a week for ten weeks. In children above twelve acetylarsen adult is administered the dosage is 1 ½-3 c c.

Whenever it is possible to give remedies intravenously then mapharside should be given according to the body weight as indicated.

Some years ago a remedy termed Avenyl an organic preparation of mercury was recommended in the treatment of leprosy complicated by syphilis but as has been indicated in a previous chapter the evidence for its effectiveness is not conclusive. The advantage of the remedy is that it dissolves in hydnocarpus oil but while one is not convinced of its efficiency in the treatment of syphilis much more work must be done before a definite statement to this effect can be made. The following is the prescription when Avenyl is used.

R Avenyl (Burroughs Wellcome)	grm 0 5
Hydnocarpus oil	100 c c
Creosote (double distilled)	0 5 c c
Dose 1-10 c c	

The method of administration is that laid down already but instead of the pure hydnocarpus oil being used hydnocarpus oil with Avenyl is given for subcutaneous injections. If Avenyl is used it is suggested it be used instead of bismuth rather than as substitute for the arsenic preparations and that a three months course be given. That is hydnocarpus oil with Avenyl be given for three months instead of hydnocarpus oil alone and in the dosage already recommended for subcutaneous injections of the oil. While we ourselves have ceased using Avenyl it is mentioned here because some authorities advocate it and further trials are necessary before any definite opinion concerning its efficacy is given.

The following principles cannot be too strongly emphasised.

- (1) Be sure the patient is suffering from syphilis
- (2) If there is unequivocal evidence of this treat along modern lines and efficiently
- (3) Give a preliminary course of six injections of bismuth before commencing mapharside

The recent work on penicillin and the treatment of syphilis is likely to revolutionise the whole therapeutics of this subject. It will however be some time before penicillin is readily available or cheap enough for it to be used in leprosy sanatoria.

B Treatment of Certain Common Concomitant Skin Affections

Because a fair proportion of cases which either seek admission to a leprosy institution or present themselves for treatment at an outpatient leprosy unit suffer from various complicating skin conditions it may be of value to indicate helpful lines of treatment. It is not the intention to endeavour to discuss the treatment of dermatological conditions in general but only to indicate practical methods of dealing with the most common skin infections. It is to be expected that persons suffering from leprosy are more prone to pyogenic, scabetic and fungus infections of the skin than the normal person. Firstly because many among those who seek admission into leprosy institutions are not in a position to attend to the routine hygiene of the skin which is of such importance in preventing skin infection and secondly the skin is often damaged because of anaesthesia resulting in disturbances of the normal sweat mechanism. Not only may the skin be affected thus but in nodular or infiltrative lepromatous leprosy many of the elements of the skin are destroyed as a result of the massive granulomatous invasion in the corium.

Further many patients suffer from a chronic avitaminosis and therefore the lack of vitamin A and vitamin B complex is liable to produce pathological conditions of the skin which not only in themselves cause lesions but increase the vulnerability of the skin to infections and hence aggravate or result in the appearance of septic and other skin manifestations. Hence in this section I shall only attempt briefly to summarise the practical methods found useful in dealing with the infective and parasitic dermatoses.

The commonest infection with which the physician in charge of a leprosy institution has to deal is scabies. Scabies in the East and particularly in India is practically always complicated by a septic condition of the skin usually a staphylococcal infection of the hair follicles. Impetiginous lesions in the form of true impetigo contagiosum are not common probably because the infecting organisms are much more commonly the staphylococci and not the streptococci. In scabetic lesions also the element of secondary infection is always prominent and one seldom comes across a case of scabies which has not a complicating septic condition of the skin hence in its treatment the physician cannot confine himself to treating the scabies alone but must attend to the pyogenic condition of the skin in general.

So far in leprosy institutions scabies has been dealt with by means of sulphur ointment. Owing to the initial difficulty of securing benzyl benzoate and the cost of the remedy it has not been tried out on any large scale. The problem which frequently arises in the management of a large number of cases is the difficulty of ensuring that any sulphur ointment prescribed is adequately administered and therefore if one of the sulphur preparations is used it must be under strict supervision. If this is possible three applications of Danish ointment will prove effective. The following is the method of preparation and technique of administration.

(1) 1 kg. of sublimated sulphur is dissolved by heating it gently in 1 kg. of a 5 per cent solution of potassium hydroxide—a clear yellow solution resulting.

(2) 225 gm. of vaseline and 225 gm. of lanoline are mixed together no heat being used.

(3) To this mixture 37.5 grm of the solution of sulphur in potassium hydroxide (see (1)) is added

(4) Fresh zinc hydroxide is prepared by mixing 28 grm of zinc sulphate and 40 grm of a 20 per cent sodium hydroxide. Then this is added to the ointment

(5) Liquid paraffin is added to make a total weight of 1 000 grm

(6) 5 grm of benzaldehyde is added to check the somewhat disagreeable smell of sulphurated hydrogen

The patient is first bathed then after the ointment has been applied he is made to sit in the sun all day. He is given a good wash at night and the process is repeated the next day. When there is an extensive infection of scabies throughout an institution the following procedure will clear it up more rapidly than any other in our experience. The patient is given a bath and if possible scrubbed and the following solution is painted all over the skin from the neck downwards giving special attention to the groins and axillae remembering scabies never affects the skin above the line of the neck.

R Sulphur	1 part
Slaked lime	1 part
Water	10 parts

Boil down to five parts and cork well

The patient is then given a bath in the evening and the procedure repeated each morning for three mornings. Then the patient is encouraged to take an oil bath for the next three or four days. It will be seen that the above treatment is drastic but as sulphur dermatitis is rare in the dark skin. For milder degrees of scabies hydrocarpus oil with 5 per cent sulphur will be found a very suitable routine remedy.

Frequently with both septic infections and fungus conditions a condition of the legs is seen which is the result of neglect and scratching the lesions become encrusted and scaly and if secondarily infected pus may form under the scales. No remedy will in such cases be of any value until the scales have separated. The following formula for Lassar's paste is recommended.

R Acid salicyl	3 I	(40 grm)
Zinc oxide	3 II	(80 grm)
Starch	3 II	(80 grm)
Vaseline	3 IV	(160 grm)

If the encrustations are thick then starch poultices may have to be applied for several days instead. Once the scales have separated then the most generally useful ointment is ung hydrog of the British Pharmacopoeia. Sometimes if the scabiness is still present this is combined with 5-10 per cent salicylic acid and if when the scales have separated a raw oozing surface has resulted then calamine lotion should be applied. If the discharge is slight the following cream will be found useful.

R Zinc oxide	grs CLXXX	(120 grm)
Sulph sublim	grs VII	(0.45 grm)
Ac carbol	℥ VIII	(0.5 ml)
Lanoline	3 4	(20 grm)
Aq calais	3 I	(30.0 ml)
Ol olive	3 I	(30.0 ml)

If the cream is not drying enough then add calamine drms II. This preparation is also excellent for prickly heat but calamine should not be added as it is

drying for this condition. If the eczematous condition is accompanied by swelling and oedema of the lower extremities raise the legs and follow the principles already laid down. Remove the crusts and soothe raw surfaces. Apply strong antiseptic or fungicide ointment only when the acute condition has subsided.

For fungus infection of the body if associated with scaling the following ointment is useful.

I	Acid salicyl	grs XXX	(0.2 gm)
	Acid benzoic	I	(0.4 ml)
	Paraff. moll	II	(0.8 ml)
	Coconut oil ad	I	(30.0 ml)

This is excellent for the common condition of Dhobie's itch. In the European it may be found necessary to reduce the strength by half. Other remedies such as chrysarobin ointment may be used but Whitfield's ointment is most generally useful.

Finally a word may be mentioned concerning the common fungus of the foot and hands (Madras foot, Bengal rot, etc.). When there are blebs and a watery discharge prior to these and apply 1 per cent gentian violet in spirit. Keep water from the part and cleanse with olive oil. When blebs have ceased then either chrysarobin ointment or Whitfield's should be prescribed. When there is much scaliness apply Lassar's paste or starch poultices then Whitfield's or chrysarobin ointment. If there is as often is the case an eczematous condition and inflammation then the tissue must be soothed first. If there is a certain amount of sepsis then preliminary dressings with warm eucal for a few days will be found beneficial. When the skin is clean then calamine lotion is applied or if not much oozing the zinc calamine cream already given and then as the acute condition clears pass on to a fungicide ointment first half strength and then full strength. It is well to remember that this condition of the foot is almost always accompanied by a fungus infection in the groin and axilla which must be treated. It is extraordinary how frequently even in Europeans the common Dhobie's itch is either neglected or not recognised and thus the feet and hands constantly become reinfected.

A common condition in institutions is a widespread infection with *trichophyton versicolor* (pityriasis versicolor). This is treated either by a 3 per cent salicylic acid and benzoic acid ointment rubbed into small areas at a time until the whole is covered or by the application of the following lotions immediately following each other.

- (a) Hypophosphate (5 per cent)
- (b) Acid tartaric (3 per cent)

No attempt has been made to cover all the skin conditions seen in a leprosy institution. Only certain points are stressed which have proved of practical value and readers when dealing with complicating dermatological conditions are advised to consult standard work on dermatology or to seek the advice of a dermatologist when available.

CHAPTER XIX

A PROGNOSIS

B CRITERIA OF DISCHARGE AND AFTER CARE

A Prognosis

The question of the prognosis in leprosy is one of considerable complexity but in assessing the ultimate outcome of the disease the question of individual resistance, invasiveness of infection, type of disease and race are taken into account. It is believed that a very fair picture of the probable course of the disease can be portrayed. In a chronic and sometimes mutilating disease such as leprosy it is essential in arriving at a prognosis to take into account not only the possibility of active disease progressing but the amount of present damage or likelihood of subsequent damage. In other words a patient with leprosy wants to know what the chances are of his complete recovery without deformity. A person who acquires leprosy has in all probability the usual concept of the disease and he imagines the day when he will become a curse to society a nuisance to himself blind and crippled and maybe mutilated. It would be very unwise to indicate to a patient that he would most certainly recover if taking every fact into account one knew that there was a great likelihood of gross deformity resulting in a few years. In other words in giving a prognosis the physician must take into account two possibilities viz

- (1) The possibility of the patient becoming an advanced lepromatous case
- (2) The possibility of deformities setting in as the result of nerve damage

If the latter eventuality is always kept in mind in patients with marked nerve involvement then the physician will advise that the greatest attention must be paid to the care of the hands and feet and suggest massage preventive exercises and all means possible to postpone such a serious end result. The prognosis of leprosy will now be discussed in the light of the introductory remarks.

With regard to the neural types in simple macular leprosy this in the case of adults is unlikely to pass into the lepromatous type but the following points must be taken into account

- (1) The age of the patient
- (2) The extent of the lesions
- (3) Clinical appearance of the lesions
- (4) The race of the patient

As has been shown only approximately 7 per cent of all children in the Silver Jubilee Clinic Sandpet in the simple macular group have become lepromatous cases during the past seven years. Hence the number in adults must be considerably less. Therefore it is safe to assume that generally speaking adults are unlikely to pass into the lepromatous stage. If however the lesions are extensive the clinical appearance indicates poor tissue immunity and especially if the lepromin is negative then a more

guarded prognosis should be given. This is particularly the case in children and young persons and in the European Mongolian and mixed races. In fact it is never safe to state categorically, for instance in the Anglo Indian with hypopigmented patches that he will never become a lepromatous case for the chances of such a person passing into the lepromatous stage are far greater than in the Indian or African races. Therefore when in doubt the patient should be advised when all signs have disappeared or when the lesions have been quiescent and active treatment has stopped to come for examination every six to twelve months.

With regard to the question of deformity or the possibility of the patches becoming permanently hypopigmented the following considerations must be taken into account: the degree of hypopigmentation and the extent of anaesthesia and nerve involvement. In other words the more pronounced the hypopigmentation the less likely is pigment to return; this especially applies to the fair person. Similarly, the more extensive the anaesthesia especially if pressure sense is impaired the greater the likelihood of permanent nerve damage and the development of trophic signs. In this connection it should be remembered that a child is more likely to get disabling deformity than an adult in the presence of extensive nerve damage. In estimating the question of permanent deformity resulting it must be borne in mind that the thin fibrosed nerve is of a serious prognostic significance as the thick and oedematous one for in the former the nerve has been reduced to a fibrotic strand while in the latter nerve destruction is not complete and there is still some chance of preventing deformity.

Neural tuberculoid leprosy of the minor degree if the lesions are extensive must be looked upon in the same light as the simple macular lesions from the point of view of prognosis. It should be added however that the more infiltration the lesions show and the more definite the nerve involvement especially in the presence of a positive lepromin the chances of such cases becoming leproma are negligible. It can also be stated that the fewer the lesions the better the prognosis. With regard to the question of residual nerve damage and its estimation this is based on the extent of the nerve involvement. Again the factor of age must always be borne in mind and a relatively closer watch should be kept on children than on adults with multiple lesions clinically of a minor tuberculoid nature especially if the lepromin is negative.

The prognosis of neural tuberculoid leprosy of the major variety is always excellent viewed from the point of view of the likelihood of lepromatous leprosy developing. It is our considered belief that true major tuberculoid cases never become lepromatous. It might be well in view of the controversy which has arisen to define what is meant by a true major tuberculoid case. A major tuberculoid case is one with grossly infiltrated well demarcated lesions with a typical histology and a strongly positive lepromin. It is our opinion that once well marked tissue immunity has been established as confirmed by biopsy findings and the lepromin test then it is not possible by any known means to break this down and the development of leproma in the case of tuberculoid leprosy would involve this.

The question of residual deformity with regard to major tuberculoid leprosy is quite another matter. Firstly because of the marked tissue immunity with histological changes extending deep into the corium permanent scarring is very likely to occur. Further there is a great probability if not certainty of permanent nerve damage with resultant severe deformity because in all major tuberculoid leprosy of an extensive nature the nerve involvement is severe. Operation and preventive exercises and electric

treatment will do much to postpone this day and in some instance stay it off for ever. It is our experience however that gross damage of the peroneal nerves and facial nerve never recovers and hence with regard to these two nerves the prognosis must be guarded. In children the prognosis of tuberculoid leprosy is similar except it must be borne in mind that residual deformity is almost a certainty. There is great scope for orthopaedic surgery in relation to nerve damage and deformity in leprosy.

We now pass on to the question of the prognosis of lepromatous leprosy. It cannot be too strongly stressed that the prognosis in lepromatous leprosy is always grave and in no case should anything but a guarded prognosis be given. In arriving at any conclusion concerning the prognosis of lepromatous leprosy the following points



FIG 138—Diffuse lepromatous leprosy



FIG 139—Illustrating the serious prognosis of lepromatous leprosy. The same patient two years later with marked infiltration of leprosy.

should always be taken into account. The younger the person the more serious is the prognosis. The longer a person has had lepromatous leprosy the graver the outlook. The prognosis is more serious if the person is a relapsed case and this is in direct proportion to the number of relapses. In early lepromatous cases one not infrequently sees recovery but after a relapse the recovery rate is much reduced and it is doubtful if recovery in the sense of freedom from disability or deformity ever occurs after the second relapse. The gravity of the prognosis also bears a direct relation to the occurrence of reactions and in this connection it may be said that whereas a reaction apart from the danger of nerve damage is favourable in the tuberculoid case in lepromatous leprosy it is never favourable for after true lepra reaction the apparent improvement in the patient's condition is deceptive and in reality the outlook is correspondingly worse. Another factor which has to be taken into account is race. The prognosis is more serious in the European, Mongolian or mixed races. Curiously enough it is one of the general

impression that fewer women tend to be discharged who have lepromatous leprosy than men. The last statement is made as a matter of interest for it is believed that it is unwise to draw any conclusions from impressions.

With regard to the question of prognosis in relationship to residual deformity, while this seldom occurs in the early case the longer the patient has had the disease the more likely will there be residual nerve damage. Nerve damage in lepromatous leprosy is due to an interstitial fibrosis and once the fibrous tissue begins to become organised and the nerve fibres are destroyed then little can be done and hence if there is not only loss of tactile sense but loss of the appreciation of pressure sense especially if it is associated with a thin fibrotic nerve then disabling deformity is almost certain to result in the course of time.

It should be borne in mind that the presence of any eye lesions is of most serious significance. With adequate eye treatment and management a great deal can be done for leprosy of the eye yet this is frequently slowly progressive and in many instances serious loss of sight and later blindness ensues due to the gross changes in the eye. Therefore the presence of eye lesions is of grave prognostic significance this is even more so if there are also lesions of the throat.

In the atypical intermediate or border line case it is extremely difficult accurately to assess the course of the disease. If in the reaction phase prognosis as to recovery is good but it is to be remembered that these atypical types are very liable to end in gross and very disabling deformities. Further there is a good deal of evidence indicating that there is a tendency for such cases to become lepromatous after a varying period hence when in doubt it is a wise rule to prognose as for leproma in all intermediate cases.

The question of prognosis in neural anæsthetic leprosy remains to be considered. In those cases who present themselves with marked deformity, bone absorption and trophic ulceration it is sometimes difficult to tell whether they are secondary neural cases or have been neural anæsthetic throughout the whole course of the disease. In both the prognosis as to deformity is hopeless for nerve destruction has already caused gross deformity. If they have been at any time lepromatous cases then there is still a possibility of relapse. While in neural anæsthetic leprosy the chances of becoming a lepromatous case are very slight it should be remembered that the lepromin test is frequently negative and hence there is always a slight possibility that such cases may develop lepromatous leprosy. In the early stages the prognosis as to deformity is based on the extent of anæsthesia and degree of nerve involvement but owing to the fact that nerve damage is usually great a cautious prognosis should be given and the patient advised to take the greatest precautions to prevent injury and if slight injuries occur to deal with them at once. In addition massage and exercises and all palliative measures for the prevention of deformity should be assiduously carried out remembering that in neural anæsthetic leprosy the chances of residual deformity are very great.

In children and young adults generally speaking the prognosis remains the same. Two points must be kept in mind in the non infective group of cases the more indefinite and numerous the lesions the greater the likelihood of the child becoming lepromatous in later life. This is especially the case in the incipient lesions of childhood or the pre lepromatous case for present investigations indicate that in a period of seven years 33½ per cent of these cases have become lepromatous. Further it must also be borne



FIG 140



FIG 141



FIG 142



FIG 143

C. The first admission in November 1940 diagnosed as an advanced proma. On 17th turn from 1st to 2nd months later the diagnosis changed. Consequently the proma diagnosis is highly improvable. Beyond not a reliable atypical action. The deceptive nature of such cases must be borne in mind when testing out alleged cures for leprosy.

in mind that where nerve tissue is grossly involved then in the growing child the likelihood of gross and disabling deformity is much greater and almost a certainty.

In conclusion while it is never justifiable to give a hopeful prognosis when one knows there are few grounds for this neither are we justified in breaking the spirit of the patient by telling him that he will not get better. In all simple macular cases and minor tuberculoid with multiple lesions in children a guarded prognosis must be given similarly in lepromatous cases it is wise to be cautious and state that at least six months must elapse before an estimation of progress can be made. Remember that all physicians who have had extensive experience in leprosy have seen remarkable recoveries even in advanced cases. While it must be admitted that the present methods of treatment leave much to be desired it can be claimed that the number of cases discharged prior to the introduction of more modern methods bears no comparison to the number discharged subsequently to this. Hope has been given to a class of persons who previously had lost all hope and in this spirit the fight should be continued and possibly some day perhaps nearer than we imagine this scourge will finally be placed among the medical problems which have become solved.

One thing is certain those adequately trained are now better able to visualise the progress of the disease and this greater knowledge leading to more accurate prognosis is a great boon to many for if it is realised that much leprosy is as innocuous as a birth mark a great weight will be taken from many shoulders and the general public will adopt a saner attitude towards this disease.

B Criteria of Discharge and After care

The question is constantly arising when should a patient be discharged from a leprosy institution or when can a patient stop treatment? In connection with the latter part of this statement and in relation to the well known fact that many lesions of leprosy are benign and need no treatment arises the question when does a case not need treatment? A maxim which should be applied until much more is known about the disease is when in doubt always treat. Unless one has had considerable experience it is unwise to withhold treatment in cases which show signs of activity. Admittedly many of these in the neural and especially tuberculoid group recover spontaneously but in a disease in which the consequences are so serious every patient has a right to treatment and only those with a really extensive knowledge can afford to refrain from treating any active case. Generally speaking however neural cases may cease treatment after they have shown no signs of activity for three months. In lepromatous cases the question is much harder to answer there is always a possibility of recrudescence of the disease and unfortunately the relapse rate is still regrettably high. It is therefore urged that a lepromatous case should be kept under observation and treatment until he qualifies for a quiescent certificate. In children who have become negative the period under treatment should be nine months. The question arises when a patient is negative should he continue to live in an infected atmosphere? There is little evidence of reinfection and for the sake of more adequate observation it is better to keep lepromatous cases in an institution until they are ready for discharge.

Should a patient continue treatment after discharge? This is another question which needs to be considered. Unfortunately there is no information on whether a person relapses more readily under treatment than without treatment. If a patient wishes treatment then small doses (6-8 c c) might be given once a week. When possible

lepromatous cases should be examined at least every three months and ideally once a month for two years. If any bacilli are discovered vigorous intradermal treatment should be reinstituted. It seems a reasonable hope that in this way there is a better chance of overcoming a relapse if it is discovered early. It must be remembered however that a relapse always increases the gravity of the prognosis.

The question of the advisability of a person marrying or not after having obtained a certificate is one which exercises the mind of many people.

(a) If the case has been a closed one there is no fundamental objection to marriage if the person is above twenty years of age and he has a quiescent certificate.

(b) It is not wise for lepromatous cases to marry as the relapse rate is so high. As this may be found to be a counsel of perfection open cases should be discouraged from marrying until they have qualified for an arrested certificate and should be watched carefully. This principle applies more strictly to women than men for the strain of child birth may be the deciding factor in a relapse.

There are many factors which will influence the physician in coming to a decision with regard to discharging a patient from his care. In institutions many cases need not be admitted because they belong to the benign neural group. On the other hand when patients come as out patients they frequently wish to carry on treatment long after the need for this has ceased. Until a more effective method of treatment is discovered this desire on the part of the patients to carry on treatment for life if necessary is understandable. While we must endeavour not to create the impression the older writers conveyed in their statement 'once a leper—always a leper' yet we must never view leprosy in a light fashion. Leprosy is a serious disease and while it is a well known fact that much of it is innocuous and benign yet a sufficiently serious attitude should be adopted which will on the one hand cause the patient who is discharged to seek advice periodically yet on the other hand not make him feel that he is in the clutches of a malignant power from which death alone will set him free.

After care of Patients

In the closing chapters of this book the need for welfare officers will be stressed. The after care of patients is an aspect which has been sadly neglected. While we cannot agree that a world should be created within a world where all who have had leprosy organize an international society with their own currency economic system etc yet Perry Burgess's (1944) suggestion contains a truth which needs emphasising. Too often patients who leave institutions are forgotten. They pass into oblivion cannot get work and in a few years time may become crippled and useless to society and a burden on the State. It is true when the disease has wrought its utmost havoc and a person is no longer able to be an effective economic unit that after care colonies should be established. These after care colonies should be able to receive all the help that orthopaedic and neuro surgery can give. Each individual should be taught some trade and learn to be a self respecting member of the community. In this connection family life should be encouraged and there is much scope for modern Papworth colonies in connection with the discharged case of leprosy. If an international organisation for the care of the arrested cases and for the preserving of the interests and welfare of the active cases were founded and if Perry Burgess's excellent ideas could be put into practice without the isolation and terror of segregation that his phrase 'a world within a world' conveys it would be a great help to those who owing to deformity or to danger of relapse

cannot be absorbed into society. It should however be emphasised that every effort should be made to encourage all discharged cases to resume their place in society unless the possibility of relapse is so great as not to warrant this risk or deformity so disabling as to prevent the patient becoming an effective economic unit. All able bodied ~~vices~~ ^{free} from infection should return to their employment without any feeling of fear or shame. Welfare officers should be attached to every institution whose task would be to follow up discharged patients help them to settle in their new environment see village town and business concerns so that work can be found for the discharged case. These officers should organise an adequate follow up and after care service. If this were done then leprosy would begin to get a fair deal and persons with leprosy would no longer feel ostracised and alone in a hostile world but would take courage and find their place again in normal society. When this was impossible owing to deformity or the grave danger of relapse then arrangements should be made for the creation of special colonies where all the resources of occupational therapy would be available and where the citizens of the colonies could make their contribution to the common good of mankind and not feel that they were solely objects of charity. This subject opens up vistas of opportunity the possibilities of which can only be imagined. It is left to men of good will initiative and determination to follow up such suggestions and create as Perry Burgess has indicated national and international groups which shall resolutely tackle this pressing and urgent problem so that the whole of leprosy may come under the purview of scientific and philanthropic men and our efforts not end at the arrest of the patient's disease.





CHAPTER XX

PREVENTION OF LEPROSY

GENERAL PRINCIPLES

From time immemorial except for a short period during the eighteenth century leprosy has been considered in some way or other an infectious disease and in almost every country stress has been placed on segregating persons with leprosy. At first every type of leprosy was isolated indiscriminately but since the Leonard Wood Memorial Conference at Manila in 1931 the principle of selective segregation has been more and more emphasised. By selective segregation we mean the isolation of infective cases from contact with healthy members of the community. This can be attempted either by compulsory or voluntary measures. There may be a number of countries in which compulsory segregation is a practical method of controlling leprosy but generally speaking the system of voluntary segregation is preferred because in any country where the leprosy problem is of great magnitude compulsory measures are financially impracticable and force the early infective cases into hiding where they are not discovered until they are no longer amenable to treatment. In the Madras Presidency for example there are probably 300 000 individuals suffering from leprosy so that if only 10 per cent of these were open cases then accommodation would have to be provided for 30 000 persons. The Government therefore has wisely decided that any form of compulsion should only be enforced as a last resort.

It is interesting to note that during the past decade there has been a very marked change in the attitude to prevention in connection with treatment as an effective measure in the control of leprosy. The report of the League of Nations at Bangkok (1930) contains this statement. In the present state of our knowledge the most important line of attack in addition to that by isolation is by treatment carried out by a trained personnel. In fact about this time the following dictum was frequently heard. Treat the early case and cure it treat the infective case and render it non infective. It was on this principle that hundreds of clinics were established throughout India. Doubts as to the efficacy of this method of control of leprosy were expressed in the Calcutta Conference of 1933. In the Cairo Conference of 1938 the main emphasis in controlling leprosy was laid on the isolation of the infective case and while treatment was mentioned it was given a subsidiary place and not considered the main weapon in the prophylaxis of leprosy. It is now almost universally accepted that treatment cannot control the disease and that the old adage just quoted no longer holds good because in many early cases the disease becomes spontaneously healed and in infective cases it takes such a long time to render a person non infectious that for the purpose of prevention such a measure is not effective.

Lowe sums up by saying that it is now generally agreed that it is hopeless to attempt to control leprosy in a community merely by establishing treatment centres.

This emphasises the fact that if an effective system of control is to be organised in any country the policy of strict and indiscriminate compulsory segregation may have to be modified on the one hand and the over emphasis of the treatment centre to the

exclusion of methods of segregation on the other. It has been well said in connection with the other human scourge tuberculosis that there was a time when tuberculosis was regarded as a disgrace and the unfortunate victim was ashamed of his affliction. Consumption somehow cast a stigma upon its victim. The social background was little understood and the public thought that people suffering from consumption were somehow blameworthy (Clark 1933). These words apply in even greater force to leprosy.

In our consideration of measures which should be adopted in the prevention of leprosy the principle contained in the above quotation must not be forgotten. Too often in the past has leprosy been considered as a social stigma and even in these days of comparative enlightenment the general tenor of many articles on leprosy is that of dealing with a social disease and the whole atmosphere is pervaded with the idea that the medical profession is doing a good but unpleasant piece of work but not related to practical medicine.

Medical and lay workers must adopt the whole view of leprosy remembering that what is seen of leprosy by many professional men is only approximately one eighth part of the whole problem while seven eighths is not appreciated because it is seldom brought prominently to the attention of the public. There are a large number of preconceived ideas which bear no relationship to facts both as to the prevalence of the disease as well as to its causation and nature. For instance while it is true that some of the highest incidences in the world can be found in the Madras Presidency of India it is equally true that there are large tracts in which leprosy is either non-existent or of such little significance that it does not constitute a major public health problem. Therefore the public health officer must not only have some knowledge as to the clinical vagaries of the disease but must possess a sane and wholesome outlook if there is to be any hope of bringing this age long endemic disease under control.

If one had time and access to the growing literature on the prevention of leprosy the tracing of development of modern preventive measures in the various countries would be of extreme interest but all that is attempted at the present time is to review the subject of prevention in the light of experience gained in India and particularly in the Madras Presidency and to lay down certain fundamental principles leaving workers to modify these according to the special circumstances obtaining in the areas in which they are working. It may however be of value to summarise the progress of anti-leprosy work in India as this affords a good example of the trend of modern development in the prevention of this disease.

In every country leprosy work was first undertaken by religious and philanthropic bodies. In the Middle Ages in England the Church was the responsible authority for segregating infected persons. In India also it has been largely due to the pioneer efforts of the Mission to Lepers and other missionary societies that public opinion has been aroused on behalf of the sufferer from leprosy.

In order to value the present work correctly we must review briefly the development of anti-leprosy work. This work in India was started in the first instance by those having a desire to help a needy section of the community but it was done at first solely on religious and philanthropic grounds with no attempt at eradicating the disease. In Europe the Church undertook the task of segregation and control and established leprosy houses outside many large towns. In India also leprosy work was first under-

taken merely as a work of compassion. Very gradually as this work was perceived to be vital to the public health there grew up the belief that institutions would ultimately bring the disease under control. The Government partly because it considered it to be a duty to look after destitute persons suffering from leprosy and partly in the hope of controlling the scourge encouraged the formation of settlements throughout India and gave grants towards their support.

As a result of this some ninety five leprosy institutions have been established in India ranging from small homes for the housing of crippled cases with little or no up to date medical work to more modern institutions accommodating 700 or 800 patients with growing facilities for dealing with all aspects of the problem. The majority of these institutions are owned and managed by the Mission to Lepers which has been the pioneer organisation in stimulating official bodies to assume greater responsibilities for the development of adequate anti leprosy work throughout India. In 1924 through the efforts of Mr. Frank Oldrieve and Sir Leonard Rogers the British Empire Leprosy Relief Association was founded. This organisation was the natural outcome of the work of Dr. Victor Heiser and Sir Leonard Rogers and others on the treatment of leprosy. The British Empire Leprosy Relief Association has as its object the stamping out of leprosy from the Empire and at that time it was thought that leprosy could be eradicated by treatment alone. For the next decade less emphasis was placed on segregation and more on the establishment of out patient clinics etc.

It was later gradually accepted that the newer methods of treatment were not producing the results anticipated and that the out patient clinic would fail to control the disease unless associated with schemes of segregation. Thus arose the idea (which has never been completely worked out) of village segregation.

At present it is felt that the work of the past has been of decided value in that (a) it has kept the problem before the public and has created an interest in leprosy and (b) the more recent propaganda through the out patient clinics has rendered the people of the country more leprosy conscious and has consequently aroused a general desire to do something for leprosy in almost every district. It is impossible to state very definitely how far either institutions or out patient clinics have helped to control the disease. Out patient clinics treating a few dozen or a few hundred cases but not connected with scientific work contribute little or nothing towards the control of leprosy. Institutions in so far as they segregate infective cases and act as centres of training and propaganda are undoubtedly of great value in the general anti leprosy campaign but apart from this it is doubtful whether they can make any adequate contribution to the eradication of leprosy unless they are linked with a comprehensive scheme of prevention.

This all shows that the present work has done much to prepare the public mind for more active steps. In so far as institutions segregate infective cases and protect others (especially children) from contracting the disease their work is of great value. Moreover the power of the example shown by the workers in institutions in caring for the destitute, the incapacitated, the helpless and the ostracised cannot be computed. It is a contribution which must enrich a nation.

It will be seen that up to 1937 as far as the Madras Presidency is concerned all that had been accomplished was to create a general interest in the problem but there had been no attempt to organise an adequate system throughout the province with one authority acting as a central control co ordinating activities and building up an

organisation which would ultimately cover the whole country. In other words if leprosy is to receive a rightful place in the scheme of preventive medicine an adequate leprosy service must be created in which officers have the same standing chances of promotion emoluments and prestige as in any other departments of the Government. The officer in charge of such a service should be a senior administrative officer with special leprosy experience and directly responsible to the head of the medical services. Only in this way can adequate attention be given to the problem and can any hope be held out to bring this disease permanently under control.

While making a plea for dealing with the whole problem of leprosy there is no desire to create the impression that leprosy should be emphasised out of all proportion to its importance as an endemic disease. If leprosy is shown to be of minor importance in a country or district then no official measure may be necessary. If as has been emphasised leprosy is looked upon as one of the endemic diseases for which preventive measures are necessary and divested of much of the false notion surrounding the subject then an adequate preliminary study of the disease will be made and measures taken which will be in proportion to its importance as an endemic disease.

As has already been indicated the work in the Madras Presidency especially during the past ten years has been developed along such lines so that the Government has gradually come to recognise its responsibility for the leprosy work and has adopted a comprehensive anti leprosy policy. In preparation for this the British Empire Leprosy Relief Association (Madras Provincial Branch) encouraged the creation of special investigation centres and as a result of this work the Government has now adopted the principle laid down in the Cairo Conference Report thus accepting the statement contained in the Government of India's Report on Leprosy and its Control in India (1942) that the development co-ordination and carrying out of a comprehensive leprosy programme is their inescapable responsibility. In order therefore to indicate methods of prevention which we feel will ultimately bring this disease under control the plan for the leprosy campaign in the Madras Presidency will be described and we hope this will provide the basis on which schemes for leprosy prevention in other countries can be developed. It must be borne in mind that any system which is established must always take into consideration local conditions as to customs education availability of adequate medical personnel etc. Principles however remain it is details which must be modified to the precise situation which is found in the country or district in which leprosy work is to be organised.

In organising an adequate leprosy campaign it is an essential prerequisite that leprosy should be looked at both from the point of view of treatment as well as prevention as an ordinary medical disease and except where special measures are necessary be treated in the wards of the general hospital. In this connection Cochrane (1944) has stated that it is of interest to note that the routine treatment of leprosy is now accepted by the authorities as a proper function of the general medical service of the Madras Presidency and that it is now agreed that if a patient has leprosy he has as much right to receive treatment at a general hospital as has a person suffering from tuberculosis cancer or syphilis. Therefore at all government and mission hospitals and at Local Fund dispensaries treatment for leprosy should be available. If the number of cases warrants it a special clinic should be organised but all clinics which are primarily for the routine treatment of the disease should be and in Madras generally speaking are under the control of the District Medical Officer (Civil Surgeon).

A further advance in the preliminary organisation of the leprosy campaign in Madras was made when an order from the Surgeon General's department was issued making it incumbent upon all district hospitals to admit patients suffering from leprosy who require immediate medical or surgical treatment either for diseases other than leprosy or for acute complications due to leprosy. It is advised that these patients should be admitted into the septic or infectious diseases wards if infective or septic and into the ordinary wards if not infective. It will take some time before the average hospital will accept this principle but not until a patient with leprosy receives the same attention as a patient with tuberculosis, syphilis or any other chronic disease will the general public be convinced that persons suffering from leprosy are any more than objects of charity and of Christian mercy. Do not let there be any misunderstanding in a well organised leprosy campaign there is this aspect of work. The Mission to Lepers and all workers in institutions which care for the crippled have set a noble example the value of which cannot be computed but unless a sense of proportion is maintained and the work related to the whole medical problem then no headway will be made.

Further before leprosy can receive the attention it merits a much greater emphasis must be placed on its teaching in Medical Colleges in countries where it is prevalent. In every teaching hospital adequately equipped and fully staffed leprosy departments should be set up and a series of lectures and practical demonstrations should be arranged. Attendance at these should be compulsory for all medical students. These lectures should ideally be not less than eight with eight demonstrations and should briefly cover all aspects of leprosy and their aim should be to endeavour to show that leprosy is a scientific branch of medicine as important and as entrancingly interesting as tuberculosis and a subject worthy of research of the highest grade. Only when medical students realise that their teachers are as keen on teaching leprosy as they are on teaching tuberculosis, syphilis or any other special branch of medicine will they take an intelligent interest in the subject. Towards the attainment of this end questions should be occasionally set in the pathology, medicine, ophthalmology and even in surgical papers. Then and then only will medical students be convinced of the importance of leprosy as an endemic disease in countries where it still remains a serious problem.

It must be borne in mind that practitioners have seldom or ever had adequate instruction in leprosy and therefore adequate post graduate courses should be planned. This all means the appointment of specialists to organise a teaching programme in leprosy but in modern medicine the services of a specialist in leprosy are as important as the services of specialists in tuberculosis, syphilis or any other disease which may be of endemic importance in a country.

It is our belief that leprosy is a disease which should be possible of control. Unless governments are willing to set aside sufficient finance and make available an adequately trained medical personnel and to establish sufficient leprosy prevention units linked to institutions and treatment centres the control of leprosy will ever remain a pious hope. It cannot be too often reiterated that half hearted measures are of little value in dealing with this disease. An age long problem such as leprosy demands the attention of the best qualified in the profession and an expenditure of money far greater than the average Provincial or Colonial Government has up to now been prepared to face.

All this means that in future leprosy workers must be paid salaries comparable to other research workers and doctors of similar grade of training and experience. It

has been realised for some time and was strongly emphasised in the Report on Leprosy and its Control in India (Central Advisory Board of Health 1942) that leprosy cannot be controlled by treatment. Therefore treatment will play only a limited part in the campaign and thus it will be understood that a leprosy policy based mainly on the establishment of out patient centres can never be successful. It should be stated lest the position be misunderstood that treatment is of value for three reasons.

(a) In certain types especially the early lepromatous variety it is believed that not only is treatment beneficial but all the evidence at present available indicates that the earlier and the more intensive the treatment the greater is the chance of recovery.

(b) By making treatment available patients are more ready to submit to the necessary preventive measures.

(c) Many cases can only be kept under observation by regular attendance at a treatment centre (Cochrane 1944).

CHAPTER XXI

PREVENTION OF LEPROSY—*continued*

With these introductory remarks we pass on to the more detailed description of measures for the prevention of leprosy. In every country where leprosy is prevalent there are institutions for the isolation and care of patients with leprosy but before the management of these can fit into an overall plan of leprosy control a great deal of preliminary work usually needs undertaking. It is generally accepted that the two most effective methods of leprosy control are

- (1) The segregation and treatment of infective or open cases
- (2) The prevention of child leprosy

Before measures can be organised on a nation wide basis however it is essential to have

- (1) Information as to the extent of the problem
- (2) Sufficient institutions of various kinds to deal with all aspects of the disease
- (3) A trained personnel both medical and lay to implement the policy laid down
- (4) Legal powers to enforce any regulations which may be considered necessary to put into effect measures adopted for the control of leprosy. We shall now discuss the 4 points in some detail

I INFORMATION AS TO THE EXTENT OF THE PROBLEM

In countries where leprosy presents a problem it may be difficult to decide where control measures should be enforced. General control measures unless the problem is a strictly limited one are not likely to succeed for it is a well known fact that leprosy is not equally serious in all districts and therefore it is only in those areas where the disease presents a public health problem that it is essential to organise extensive control work. I indicated as far back as 1933 in a report to the Government of Ceylon that information concerning the number of cases of leprosy without details with regard to age type etc. might give no adequate information and cause health authorities to concentrate on the wrong areas.

Reviewing the leprosy situation as a whole it will be found that there are areas sometimes villages contiguous to other highly infected areas or villages in which the incidence of leprosy is negligible. On the other hand there may be areas where leprosy may be prevalent but is not serious enough to warrant taking active measures whereas there may be still other areas or villages where not only is the incidence of the disease high but leprosy is actively spreading. In a country with a high leprosy endemicity it is manifestly impracticable to deal with the whole problem and therefore it is contended that by concentrating measures on areas where the disease is actually spreading leprosy will be brought under control more quickly than by trying to cope with

the problem as a whole. This raises the question how is it possible to find these areas?

In every country where leprosy is a serious problem there should be appointed a medical specialist to direct the leprosy campaign. The director of the leprosy campaign should be responsible to the head of the medical services and it is he who would ordinarily advise the Government as to the areas in which active anti leprosy measures are necessary. This means that attached to the office of the leprosy campaign should be a survey personnel who would under the director gather together all information concerning the existence of leprosy in a given district and proceed to alleged highly endemic areas to investigate whether active measures need to be taken.

Leprosy is usually so widespread in countries where it is prevalent that it is impossible to undertake measures of relief which will deal with every district or area where there are cases. Even in a single district only such cases as are an actual or potential danger to the public health can be dealt with and therefore it is essential to be able to estimate whether the problem in a given locality is serious or not. It is therefore important to gather information about the general incidence in a district prior to the organisation of more intensive surveys and preparatory to the development of more detailed plans for bringing leprosy under control. A very general idea of the distribution of leprosy can be acquired by the following methods:

- (a) Examination of school children
- (b) Examination of registers in out patient clinics
- (c) Examination of representative groups in a given area

(a) Examination of School Children

Where school attendance is either compulsory or where the population are school conscious then this is probably the most practical method of estimating the relative importance of leprosy in a district. In the first place it is comparatively easy to ensure the examination of a large percentage of children. In this connection it is better to train the ordinary school medical officer to undertake this examination at a time when the routine physical examination of children is being conducted rather than arrange a special examination for when this is done there is a tendency for parents to persuade their children whom they know to be suffering from leprosy to absent themselves. Further if this is done leprosy is related to the other endemic diseases liable to occur in a school and not specially emphasised. Such an approach helps to bring the disease in the eyes of the teacher and public into its right perspective. The name and address of all children should be taken and whenever cases of leprosy are discovered the school medical officer or an adequately trained assistant should make arrangements to visit the homes of the children so detected.

(b) Examination of Registers in Out patient Clinics

While out patient centres by themselves as stated contribute little or nothing to the control of leprosy yet useful information can be gathered from an examination of their records for they should show the number of cases which have been registered over the years their type and the village or street from which they come. In this way a comparison can be made of the relative intensity of infection between one village

and the next. Unfortunately except in cases where the doctor in charge of a clinic happens to have enough experience the record of the type of case attending the clinic is not as a rule accurate enough on which to pass any judgment with regard to the relative numbers of each type coming from the areas served by the clinic. Nevertheless the majority of out patient clinics in India give sufficient information to serve as a comparison between the number of cases coming from the various areas rural or urban in the district.

(c) Examination of Representative Groups in a Given Area

Where recruits to the army or police force are examined valuable information can be obtained provided the medical officer is sufficiently interested and trained to recognise the disease for generally speaking recruits come from all over the country and it is reasonable to suppose that if leprosy were endemic in a given area a larger proportion of recruits from such a locality would be discovered to have leprosy. This method is of limited value for as the result of experience in the war (1939-43) it was remarkable how frequently early cases were missed among recruits.

Sometimes if leprosy is reported to be prevalent in a certain area by making friends with the headman or village munsiff a preliminary investigation can be undertaken by an examination of the crowd which always gathers round when a fresh visitor arrives. This examination should start with the more friendly disposed persons and gradually the majority of the crowd will join in the fun of the game. Every area roughly surveyed should be marked on a map so that the distribution of open and closed cases can be seen at a glance. If preliminary information of the incidence of leprosy is gathered in this fashion gradually the rough outline of the distribution of leprosy in a given area will shape itself. The next stage in the campaign is to establish one or more survey parties to undertake the intensive survey of villages in each district in which leprosy is prevalent. For instance in the Madras Presidency apart from the suggested field personnel attached to the director's office there will ultimately be formed ten survey parties whose headquarters will be in the headquarters town of the district and whose responsibility will be to investigate all information indicating that a given town or rural area has a high incidence of leprosy. Admittedly in a country like India if such a policy were developed for the whole country a very large organisation would be necessary but each province is so large a territory that it must be considered separately and if leprosy is to be adequately dealt with there is no other way by which to secure the necessary information. The following is the minimum personnel required for a survey party.

One field investigation medical officer

One clerk

A nurse or health visitor—it is essential that this person be a woman

A peon or messenger

The object of an intensive survey is to obtain as exact information as possible concerning the state of the epidemic of leprosy in a given district in order that it can be decided whether active measures are needed to control the infection. As yet there has not been worked out a leprosy index by which the situation may be gauged at a glance. As a result however of continuous field investigation it is hoped that the intensity of infection may ultimately be represented by a standard formula so that one area can be easily compared with another.

At present we consider the following criteria are of maximum importance in determining whether leprosy is a serious endemic disease

- (1) Gross incidence i.e. number of cases of leprosy per thousand of the population examined
- (2) Child incidence i.e. number of children with leprosy per thousand children examined
- (3) Open case rate i.e. number of open cases per hundred cases of leprosy
- (4) Child rate i.e. number of children with leprosy per hundred cases of leprosy
- (5) Sex ratio i.e. ratio of male to female cases in the area based on the sex incidence (number of male and female cases per thousand of the population)
- (6) Age group distribution i.e. number of cases in the various age groups (0-14 15-34 35 and above)

It can be stated until more accurate information is acquired that if the gross incidence in the Madras Presidency is above 30 per thousand and if the child rate is 30 per cent or above and the open case rate 25 per cent or above the situation may be serious.¹ This opinion we think would tend to be confirmed if in addition the ratio of males to females had altered from the usual three or four males to every one female to two males or even less to every female infected and further if the distribution of the cases in the various age groups showed a greater percentage of cases in the 0-14 and 15-34 age groups than in those above 34. It is not the number of cases in a given area that necessarily matters it is the percentage of open cases and child cases linked with the age group distribution and the sex ratio that appear of greater importance. It is manifestly impossible where leprosy is widespread to undertake measures of active prevention in every area. Therefore the areas of greatest importance are first chosen and as the disease is controlled in those areas or villages the areas of less importance are subsequently dealt with; this means the constant utilisation of the survey parties which can be looked upon as active patrols continuously investigating the enemy's position so that headquarters can so dispose of available forces so as to check enemy movement wherever it becomes active. All this involves reasonable accuracy in survey methods and necessitates the examination of every man, woman and child in the area chosen for intensive survey. Details of methods of survey have been laid down in the Report of the International Congress of Leprosy held in Cairo (1938) as well as in the Report on Survey published by the Indian Research Fund Association (1941). Those organising anti leprosy work are advised to consult these publications.

The pioneer in survey work in India is Dr Isaac Santra whose detailed methods have been largely adopted for the Madras Presidency. In the appendix to this chapter several sample surveys are given to indicate the method adopted and to illustrate the basis on which conclusions can be drawn.

A word should be added with regard to the scheme originated by Muir (1929) designated by the name PTS—Propaganda Treatment Survey—and reasons given why it has not been recommended. The plan consisted of a survey party visiting an area carrying on propaganda establishing a treatment centre and simultaneously undertaking a survey. This method has several disadvantages. (1) In the first place

¹ The incidence and proportion of the various types of cases and of child infection vary so greatly that officers in other countries will of necessity have to make their own estimation as to the relative significance of the incidences and ratios in the areas surveyed.

propaganda is largely based on the effectiveness of treatment—this is considered unsound. (2) The establishment of treatment centres gives support to the older conception that leprosy can be controlled by treatment. (3) When the survey party leaves no permanent preventive scheme is left and the treatment centre gradually loses its popularity and any good that it may have done tends to be nullified by the subsequent disappointment consequent on the failure of treatment as a whole.

II SUFFICIENT INSTITUTIONS OF VARIOUS KINDS TO DEAL WITH ALL ASPECTS OF THE DISEASE

It cannot be too strongly emphasised that without well organised institutions a balanced system of leprosy control is impossible to develop. Institutions of various



FIG. 144—Main entrance Lady Willington Leprosy Sanatorium
(Photo by E. L. R. H. E. J.)

kinds will always have a place in the leprosy campaign and it is hoped that the day when organised institutional work is considered superfluous has long since passed. Such a viewpoint may have been understandable in the first flush of enthusiasm on the introduction of the newer methods of treatment but now that the work of the past twenty years can be more accurately appraised there is no justification for adopting such a point of view. Institutional work will be considered under the following heads:

- (1) Leprosy sanatoria
- (2) Leprosy colonies
- (3) Children's sanatoria
 - (a) For children with leprosy
 - (b) For healthy children whose parent or parents have leprosy
- (4) Homes for advanced and crippled cases
- (5) After care colonies

(1) Leprosy Sanatoria

The following description of the organisation of a leprosy sanatorium has been kindly furnished by Dr Donald Dow, lately medical superintendent of the large leprosy hospital founded by the late Dr Isabel Kerr at Dichpali in the Nizam's Dominions, South India.

A modern leprosy institution should be a combination of hospital and sanatorium, i.e. it should have hospital accommodation for patients suffering from complications of leprosy or co-incidental diseases and it should have cottage accommodation for patients who are not bed-ridden and who can do some form of work.

Such institutions should not be in proximity to towns. They should have sufficient space to allow of development and avoid overcrowding and there should be a considerable area of land devoted to agriculture. Water supply should be adequate. Leprosy sanatoria should not be so small that they lack an all-round life and are not economical nor should they be so large that all personal touch is lost. Admissions should be on a voluntary basis¹ and apart from neural cases which may require some special form of treatment (operation, electricity, etc.) the patients should generally be composed of lepromatous cases.

Buildings

These should include ward accommodation, operating theatre, buildings for routine and special treatments, laboratory—these are the hospital buildings. The residential accommodation should consist of cottages which are well ventilated and so constructed that they can be kept clean—the quarters for men, women and children can be located in different parts of the compound. Administrative block—this will vary in size according to the size of the institution. Buildings required for farm work, educational and industrial activities and for concerts, meetings, etc. There should also be adequate sanitary arrangements.

Food

It is essential that patients be supplied with a nutritious diet and the practice of supplying grain and a cash payment to purchase the additional food stuffs required is not satisfactory. The diet should be under medical supervision and should satisfy the demands of nutritional experts. The most satisfactory way of doing this is to issue food from a central kitchen as is done in general hospitals. An institutional farm can be of great help in supplying vegetables, fruit, milk, grain, etc. for kitchen use.

Labour

Occupational therapy is a very important adjunct of medical treatment in leprosy and the type of work should be useful to the institution and according to the capacity of the patients. Many forms of work can find a place in the organisation and they will vary according to the size of the institution and its locality, so the following list is only tentative. The various sections are controlled by a technical expert on the

¹ The principle of voluntary admission is accepted except under special circumstances—these are indicated later.—R. L. C.

PREVENTION OF TYPHOID

testimony but the work is performed by agents in charge of an elder (patient) and is responsible for seeing that the expert's orders are carried out.

- (1) Farm Field and garden work
(2) Acker Cooking for women attending to animals
(3) Hospital Older boys and girls can work in wards and give injections
(4) Education Men in my series as carpenters masons builders etc
(5) General Men can clean roads do anti malarial work etc Women can wash and mend children's clothes



(The signature is at the end)

P.O. Box 100 - New York, N.Y.

Do not want should be made for about patient's health & it is illegal

Social Life

3. sanatorium is a very self-contained unit and provision should be made for (a) games and sports (b) concerts and dramas (c) lecture hall and cinema shows (d) meetings and services

Law and Order

It is very essential that discipline should be maintained but it makes for greater happiness and contentment if the parents feel that it is not an imposition from above but something in which they have a share. This may be accomplished by having

- (1) *Periodic Election of Elders* They should hold office for a limited period and

be responsible for seeing that their living quarters are clean and tidy that patients regularly attend for work and do the allotted tasks that irregularities are reported for trial and penalty

(b) *Regular Courts for Trial* The court should consist of a chairman who is a member of staff (e.g. teacher) and there should be a jury of elders who will not only return a verdict but give their ideas on punishment. This system is very satisfactory in practice and means that the administrative head of the institution can content himself with occasionally reading minutes of trials and seeing that there are no miscarriages of justice.

Staffing

We have left a consideration of staff until the end not because it is of minor importance but because by first describing the various activities of a leprosy colony it will be evident that a team of workers with some sense of vocation is required. Success or failure is largely dependent on the spirit in which the work is done and it must be remembered that patients are frequently suffering from a psychic trauma which demands sympathy and understanding. The following is an outline of the staff required and their responsibilities.

Medical Superintendent As in mental hospitals and tuberculosis sanatoria the administrative head should be a doctor. He should be responsible for

- (a) carrying out the policy of the Government Department or Committee of Management
- (b) seeing that all members of staff perform their duties
- (c) discipline of staff and patients
- (d) allocation of duties to subordinate medical staff
- (e) directing medical work also educational work in leprosy for doctors and students
- (f) advising authorities on leprosy problems

Business Manager He is the right hand man of the superintendent and should undertake

- (a) responsibility for office routine
- (b) see to buildings and repair work
- (c) have supervision of work of various technical experts

Medical Officers They will carry out duties as directed by the medical superintendent.

Nursing Sisters They should have oversight of ward work, injections, operations, linen and all that pertains to the duties, activities and responsibilities of a nursing sister.

Teachers, Agriculturist, Tradesmen, etc. They have the direct oversight of the work in school, farm, etc. and report as required to the business manager.

Regular staff meetings should be held and reports made and suggestions received. In this way the unity of the institution is achieved and the co-operative spirit which makes for smooth running maintained.

(2) Leprosy Colonies

In countries in which more primitive conditions exist, leprosy institutions can be organised on the system of a colony. This is particularly suitable in Africa where

whole villages are organised in the form of a colony and where patients live as normal lives as circumstances permit. In such colonies patients live in huts and there are special hospitals and infirmaries designed for those who need medical care or who are crippled. Under such a system and in more primitive countries it should be possible to make a colony at least as far as food is concerned self supporting. It might be said in passing that it is doubtful whether a properly organised fully staffed and equipped colony could ever be truly self supporting and therefore agricultural and other pursuits should be looked upon as forms of occupational therapy helping to provide all or part of the food and keeping the patients happy and contented. Much work has been done in the organisation of large colonies especially in Africa where the system



FIG 146.—Panoramic view of the L. dy Willingdon Lep. sy Sanat. um, Chingleput, South India.

of clans and headmen lends itself to such attempts at control. The question of marriage must be considered in the organisation of colonies on the clan or tribal basis for unless this is permitted it would be impossible to consider such a method of control. In leprosy sanatoria and hospitals so long as compulsory segregation is not enforced and patients present themselves voluntarily it is well better to discourage marriage than is particularly the case under Indian conditions.

(3) Children's Sanatoria

- (a) For children with leprosy
- (b) For healthy children whose parent or parents have leprosy

(a) For Children with Leprosy

With the increasing emphasis on leprosy in children the question of sanatoria and other types of institutions for child leprosy is receiving greater attention. When

the question of methods for the control of rural leprosy is discussed in the concluding chapter it will be mentioned that it is impossible to segregate children under village circumstances and therefore it is a prime necessity to send all children with infective or potentially infective leprosy including those who have or are likely to develop gross nerve damage to a children's institution. Such a sanatorium may be part of an existing institution or a separate institution altogether as for example the Children's Leprosy Sanatorium in Ettapur Salem District South India. In the organisation of such an institution the policy with regard to admissions should be carefully thought out. All children who are infective or potentially infective should receive preferential admission. Early benign lesions likely to retrogress in the ordinary course of the disease should



FIG. 14.—General view of hospital Lady Willington Leprosy Sanatorium
(The M.B.L.P. Reg.)

be kept under observation in the environment in which they live for unless it is essential to admit a child either because it is infective potentially infective or in danger of becoming deformed or actually has nerve damage then it is far better to permit him to carry on under normal circumstances for only in this way can a right appreciation of the disease be conveyed to the general public. Under these circumstances it will be seen that children's sanatoria should be thoroughly equipped to teach children and to train them to be useful citizens. Unfortunately lepromatous leprosy in children is of serious prognostic importance. There is a grave likelihood of a child passing into adult life with lepromatous leprosy. When a child has reached the age of sixteen he should be transferred if still infective to an adult institution.

The child crippled with leprosy is a most pathetic figure and therefore in all children's sanatoria a section should be set aside for such cases and this section should have specially trained personnel to deal with orthopaedic surgery occupational therapy and physiotherapy and in addition there should be a rehabilitation centre. As far

as is known no child leprosy sanatorium is so completely equipped as this. The child is the most precious heritage of the nation and to us who know the wastage of life caused by leprosy the sadness of the little one who has fallen by the way on account of leprosy comes as a challenge to endeavour to see that no child suffers acute physical or mental torture through the ravages of leprosy. A vista of enormous possibilities opens up for child leprosy awaiting the pioneer who will answer the cry of the child who through no fault of his own has acquired leprosy.

(b) *For Healthy Children whose Parent or Parents have Leprosy*

Generally speaking all healthy children should be brought up in their own environ-



FIG 149.—Mun ho pital ward, Leeds Wellington Leprosy Sanatorium.
(Phot by B. L. K. H. E. G.)



ment and not in an artificial atmosphere. For if they are placed in such an institution they become divorced from the life of their community and are liable to become institutionally minded with the resultant psychological problems which tend to arise in children who have become homeless. Nevertheless it is a fundamental axiom that the children of parents with infective leprosy should be separated preferably at birth and certainly at as early an age as possible from their infective parents. It is more reasonable to remove the source of infection to an isolation centre than remove the child and in India this frequently can be done for there are usually relatives able to care for the child. While the segregation of healthy children of parents with leprosy has been rightly stressed it must be remembered that possibly as many children acquire leprosy from relatives other than parents than do the children of the infective case. On page 21 a table is given showing that children in South India are infected from sources other than their parents more frequently than from the parents themselves and therefore not only the healthy child of parents with leprosy must be taken into account but all



FIG 10—Occupational therapy—weaving
(Phot. by P. F. R. H. F. G.)

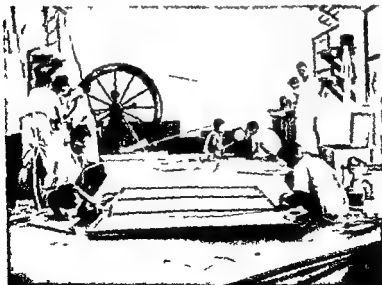


FIG 151—Occupational therapy—repent y
(Phot. by R. L. F. H. F. G.)

revised. In fact it is our belief that lepromatous leprosy does not unduly shorten life for the expectation of life is frequently as high as that for the country in general. If this then be the case the time has come to consider the question of infirmaries for such cases. All institutions for the isolation of leprosy should include in their plan an infirmary section. The nursing and care of the advanced case is a constant tax on the staff of the institution. These infirmaries should be separate from the main institution but should be an integral part of it and special arrangements made to facilitate the nursing of such cases. A suitable staff and equipment should be provided for the care of the bed ridden. In such infirmaries proper bathing, cooking and toilet facilities should be available. Apart from the humanitarian side of the work there is a great deal of investigation yet to be done in advanced leprosy. The arrangements



FIG. 15 —Social activities. Patients acting an Indian drama.
(Photo by F. I. R. M. Fsq.)

for caring for the deformed and advanced case lag far behind the need and no modern state should tolerate the position now seen in some countries.

The beggar with leprosy should not be specially selected for discriminate treatment. This situation can only be met by approaching the problem of mendicancy as a whole. This is largely an urban problem which has never been taken up in a comprehensive manner. As far as possible beggars should not be treated as criminals. Where there is evidence that an individual or individuals are making their livelihood out of this unfortunate class of person then they should be severely punished. Arrangements should be made for the care and permanent housing of all persons whose economic condition is such that they cannot maintain themselves without resource to begging. Institutions of this kind should be staffed and managed by a philanthropic organisation which is experienced in this work. If facilities for the voluntary care of these persons are available then all those who refuse to isolate themselves in such homes or escape

should then be treated as trespassers against the law and be incarcerated in a prison. In these beggar homes facilities should be given for the separation of infective cases of leprosy but no person with leprosy who is non infective need be specially separated from the general class of beggars.

(5) After care Colonies

This is an aspect of the work which should receive increasing attention. While the general principle should be adopted that persons who have recovered from leprosy should under normal circumstances be reabsorbed into society and every effort should be made to emphasise that this is to be expected there will always be a certain number of persons who may find that it is impossible to earn an economic wage and by virtue of the fact that responsibility has been accepted by the authorities through their admission to a leprosy institution provision must be made for their future care. This specially applies to those cases which are deformed or are likely to have a serious relapse if returned to an unfavourable environment. The creation of after care colonies would open up the way for employing young people who had been brought up in homes for healthy children or who had been discharged from children's leprosy sanatoria.

III TRAINED PERSONNEL BOTH LAY AND MEDICAL, TO IMPLEMENT THE LEPROSY CONTROL PROGRAMME

In the concluding chapter detailed methods of prevention in rural and urban areas and the relationship between the several units and the whole control organisation will be indicated but before the complete leprosy control service is established an adequate number of trained workers both lay and medical must be available and therefore the matter of the organisation of training courses not only for the specialist but also for lay workers must be considered.

One of the greatest handicaps in the organisation of the leprosy campaign is the lack of realisation among the medical profession that leprosy is primarily a medical and public health problem and secondarily a social one. While directors of medical and sanitary services are fully cognisant of the necessity for undertaking active control measures in malaria, plague, sleeping sickness and other communicable diseases generally speaking their approach to the leprosy problem is elementary in the extreme. The reason being that in schools of tropical medicine the modern aspects of leprosy are not given sufficient emphasis. It is therefore imperative that training in leprosy should start in the medical school in countries where leprosy is prevalent and the whole course of training in schools of tropical medicine should be drastically overhauled. Cochrane (1944) states as follows:

Every medical college in areas where leprosy is an important endemic disease should have a separate leprosy department in the charge of a physician who has higher medical qualification. The department should work in close association with that of dermatology but leprosy should not be considered merely as part of dermatology for then it would be given scant attention. Where leprosy is not an important disease in a province then it may be considered along with dermatology but the lecturer should himself have taken an advanced course in training. The prevailing opinion that a fortnight's course in leprosy qualifies a person to become a

leprosy expert should not be encouraged. A disease which has baffled mankind for centuries cannot be mastered in fourteen days.

Post graduate teaching in leprosy should cover the following courses

- (1) Elementary (2) Advanced (3) Refresher (4) Specialist

(1) Elementary Courses

These need not be for more than fourteen days duration and all practitioners should be encouraged to take at least this course.

(2) Advanced Courses

These should be open to all who have taken the elementary course and wish to have a fuller acquaintance with the subject and should be of a month's duration. All persons in charge of out patient leprosy work should be encouraged to take this course as a very minimum.

(3) Refresher Courses

These should be for senior medical officers, health officers and school medical officers and of ten days duration. The object of such an abridged course is not to train specialists but to give district medical officers and those in administrative positions up to date information so that they may be able to take a greater interest in the treatment and prevention of leprosy. Too often a leprosy clinic is established and a junior doctor is placed in charge and the medical head of the district is unable to encourage or help because he knows very little about the subject. If such refresher courses as indicated were available the doctor in charge of the routine treatment of leprosy would receive encouragement and help and leprosy treatment would become an integral part of the out patient department of a district hospital and not relegated to a shed in the back of the compound and given the minimum and scantiest attention. Health officers should be encouraged to take this course so that they too may be in a better position to co-operate in any leprosy campaign.

(4) Specialist Courses

These should be for those who intend to specialise in leprosy and should be of six months duration at least. The course should cover all aspects of the problem including the practical working and management of an institution. Such a comprehensive course should be taken by those who are in charge of leprosy institutions, survey units, teaching departments and any special investigation units. Only thus will men of adequate calibre be attracted.

Courses for Lay Workers

In the development of the leprosy campaign the lay worker must not be left out of consideration. His value has been demonstrated in the many laymen who are superintendents of leprosy homes and in Africa where under the auspices of the H. M. S. lay workers have done invaluable work. Attached to every institution and to the main leprosy departments of colleges and headquarters hospitals should be one or more lay workers whose duties would be to take a personal interest in all patients, study their environment, encourage them to persist in treatment and advise them to take precautions if infective. He would also endeavour to find means of assisting the family

if the bread winner had to be admitted into a leprosy sanatorium and follow up contacts and discharged cases endeavouring to see how the discharged case is standing up to work outside and how he is being received into the general community. There are innumerable ways the lay worker could help to bridge that gap which is at present altogether too great between the doctor who is doing what he can medically and the lay public or patient who through ignorance folly or deliberate carelessness undoes all the work which is being built up not only to create a right attitude towards leprosy but to help to stop the ravages of a disease which has blighted the lives of mankind for centuries and only now is beginning to receive rightful attention. To some it may appear that we are overstating the facts but inquiry at any out patient leprosy department or taking a random selection of patients in any sanatorium will reveal a grim tale of sadness the like of which is difficult to find elsewhere. It is the gaunt spectre of prejudice persecution and fear which must be laid low. It is to the educated enthusiastic lay worker the challenge is issued for it is he who can best convince and help the public to understand that leprosy is not a visitation of the gods but as all disease an accident of life that human beings are not merely outcasts objects of pity and compassion but ordinary citizens many of whom can still carry on others of whom should be protected and cared for not as if they were unclean but as honourable casualties in the grim battle against disease. To those who hear the call to work as welfare officers and lay workers there is no George Medal or Victoria Cross but the satisfaction that their fellows shall be saved from blight and stigma and helped to raise their heads among men as those who are not ashamed but are honoured as soldiers are honoured who have fought well and bravely and have become severe casualties in the battle against disease. One day in God's own time we shall celebrate V.L. day (VICTORY OVER LEPROSY) but till then let us not grow weary but pursue this warfare until not only leprosy as a disease is controlled but all the horror and unreasoned fear that accompanies it is banished for ever from the mind of man.

IV LEGAL POWERS

In any comprehensive anti leprosy scheme the part legal powers should play in helping to enforce measures needed to be devised for the prevention of infection should be considered. A fundamental principle must be laid down that while regulations at times have to be devised for the protection of healthy members of the public especially children on no account must the force of law be used so that sufferers from leprosy are branded as criminals and worthy of punishment. One only needs to compare the attitude towards the mentally afflicted to day and twenty or thirty years ago and if the legal aspect in leprosy is reformed to the same extent as that in mental disease a great step forward will have been taken. In this connection it should be stated that it is not only unnecessary but unjustifiable and cruel to institute legal powers against all cases of leprosy irrespective of the type. Secondly it creates a great sense of frustration if those who can protect themselves from infecting others—and as will be seen the precautions are very simple—and have power to do so are summarily incarcerated in a leprosy institution without being given the chance to see whether they are willing to play the game and act the part of good citizens. In a country where the leprosy problem is limited and where there is the possibility of bringing every open case under segregation compulsory segregation may be justified but as in mental illness those

who need isolation as a protective measure should be given (a) the chance of seeking voluntary admission or (b) where facilities are available of isolating themselves in their own homes. Only when a patient cannot or will not listen to persuasion should the force of the law be invoked. In other words all measures with regard to compulsory segregation should be as the British policeman's baton hidden only to be used in case of an emergency.

Another principle which should be enunciated is that no closed case of leprosy needs isolating from healthy members of the community. If the patient is crippled or has ulcers which need care then he should be admitted into a home and cared for but not treated as a criminal and forcibly isolated. It is doubtful whether blanket regulations are ever justified in dealing with leprosy for another axiom which needs to be reiterated is that measures taken for the isolation of infective cases must keep pace with available accommodation. In order to illustrate the development of the legal aspect of leprosy the Madras Public Health Act (1939) is an excellent example. When this Act was first promulgated no person with leprosy, no matter whether it was infective or non-infective, was allowed to appear in a public place to travel by train, bus, horse or man-drawn vehicle or to do anything which involved his coming into contact, slight or intimate, with anyone outside his own house. There were however no provisions made to prevent the patient from staying at home and infecting all his children. In consequence the Act, as far as leprosy was concerned, was observed in its breach rather than in its compliance. It was therefore found necessary to amend it drastically. As an appendix to this chapter the present provisions for leprosy in the Public Health Act are given. It will be noted that in the first place leprosy is defined as that type which is open and from which bacilli can be discovered from the skin or mucous membrane of the nose by standard methods of examination. Secondly, the force of law is only resorted to after an area is declared a segregation area and sufficient facilities are available for the isolation of all infective cases. This means that legal powers must keep pace with facilities for segregation and that only areas where leprosy is an important endemic disease need be declared areas under segregation. The position with regard to this is briefly that if a group of villages are found to be infected and facilities are organised for the isolation of open cases then these villages can be declared segregation areas under the Act. If a patient does not submit himself to the regulations concerning the segregation of open cases he can be made to do so under the power of the law. The declaration of certain areas as special areas does not appear to be of much value apart from facilities for segregation. Generally speaking widespread measures to prevent patients with leprosy, whether open or closed, travelling in public conveyances, using town or village water supplies, buying food in the bazaar, etc., defeat their own end for they are impossible of enforcement. In other words only as facilities for segregation are available and special measures to deal with infective cases devised are any regulations prohibiting employment, travelling, etc., likely to be successful.

Too often leprosy regulations penalise the poor and the outcast and leave the rich and influential unaffected. Further, only when a realistic view is taken of leprosy and the whole campaign is related to the principle of controlling leprosy in areas where it is spreading, can there ever be any likelihood of the development of a system which will receive the support of the public and be calculated to arouse the interest of the profession and achieve the required result, namely the elimination of leprosy from the country in question.

CHAPTER XVIII

PRACTICAL METHODS OF LEPROSY CONTROL (as illustrated by the proposed organisation of the Leprosy Campaign in the Madras Presidency)

THE Madras Presidency of South India represents an area where it is possible to embark on a leprosy prevention programme on a wide scale. The outline of the work already done and a description of the possible developments will illustrate the methods along lines which we believe will most effectively bring the disease under control.

In January 1945 the preliminary building up of the leprosy organisation received official approval by the creation of a special appointment by the Government of Madras of a Director of the Leprosy Campaign. The duties of this official were combined with those of Director of Leprosy Research and the main task of this officer was to correlate all the official leprosy work in the Presidency and gradually build up an organisation which would cover

- (1) Rural leprosy
- (2) Urban leprosy
- (3) Child leprosy
- (4) Research
- (5) Teaching
- (6) The relationship of existing and new institutions to the general leprosy campaign
- (7) The place of the general hospital and out patient clinic in the campaign
- (8) Propaganda



FIG. 153—General view Rural Leprosy Preventive Unit

(1) Rural Leprosy

For many years ago emphasised the fact that leprosy was largely a rural disease and that while there was a considerable amount of leprosy in the larger towns in districts in which the disease is prevalent yet there was a constant migration for various reasons—economic, social, industrial—from the villages into towns. Occasionally there is a reverse movement as reported by Chandy in the Fyzabad district of the United Provinces where persons who had migrated to Burma have returned to their village

with leprosy and set up foci of the disease. It is our firm belief that leprosy can only be controlled in rural districts by the establishment of rural preventive units and to this end the first rural preventive unit was begun in the Madras Presidency in 1939. The object of these units is to deal with leprosy in those rural areas where the disease is serious. It is manifestly impossible owing to the enormous expense involved to provide complete segregation for all infective cases neither do we think that this is necessary for we believe our task is to discover the minimum amount of segregation which will control the disease. In the Middle Ages in Britain as already pointed out it was evident that complete segregation was not attempted. The regulations which were enforced through the authority of the Church resulted in certain precautions being taken in the day time and absolute isolation at night from all healthy persons.



FIG. 154.—Segregation huts

particularly children. It is well known that in India at least the majority of villagers are agriculturists and therefore are in the fields the greater part of the day. It was thought therefore worth while commencing an experiment in leprosy control in a group of villages in the vicinity of Chingleput. At first nine villages were chosen but this was found to be too large for practical purposes hence the experiment was cut down to four villages with an approximate total population of 3,400 and an incidence of leprosy varying between 78 per thousand and 29 per thousand.

Two plots of land were selected within two miles of the villages surveyed and on one administrative treatment and staff blocks have been built. These consist of

- | | |
|---------------------------------|-----------------------------|
| (a) Doctor's house | (e) Nursing orderly's house |
| (b) Store house and water tower | (f) Gardener peon's house |
| (c) Well and pump | (g) Laboratory |
| (d) Compounder's house | (h) Treatment centre |
| | (i) Guest house |

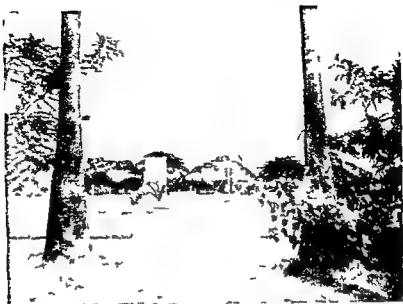


FIG 1 —Staff are — how: water ton and om of taff hou Not they
re b It in simple ill go tyle w th thatel ed roofs



FIG 156 —Lab t ry an l tre tm at hall

On the other side of the road the segregation area is built which consists of seven houses and a well. The whole capital outlay including equipment was Rs 8382/6/ (approximately £650) and the annual recurrent cost is Rs 2230/ (approximately £170).

An intensive survey has been completed and for the past five years all open cases from the four selected villages have been required to sleep in the segregation area at night thus ensuring the prevention of night contact with children. Because of the difficulty of dealing with women and children who are open cases these are persuaded to seek admission into the Lady Willingdon Leprosy Sanatorium (twenty three miles away). To all who come to sleep at night a ration of rice is allowed sufficient for an evening meal. One of the philanthropic minded gentlemen in a near by village has been contributing rice for this purpose and must have saved the scheme over the past five years thousands of rupees.

The experiment in night segregation has continued for five years and the following table gives the comparison and summary of follow up surveys in these villages over this period.

SUMMARY OF SURVEY FOR THE YEARS 1939 1942 AND 1945 OF THE
RURAL INVESTIGATION CENTRE MADURANTAKAM

Village	1939			1942			1945		
	Gross Incidence	Open case Rate	Child Rate	Gross Incidence	Open case Rate	Child Rate	Gross Incidence	Open case Rate	Child Rate
*Polambakkam village	42.52	25.80	32.60	52.89	23.21	20.58	44.13	21.87	9.37
*Polambakkam cheri	45.05	23.80	19.05	44.00	13.63	13.63	38.70	16.68	16.66
*Perambakkam village	38.46	28.57	14.28	38.64	31.25	18.75	28.39	18.18	9.09
*Perambakkam cheri	78.12	30.00	50.00	60.60	37.50	50.00	82.19	33.33	20.00
Maluvankaranai village	33.12	45.45	18.18	48.41	29.41	23.53	70.60	19.23	23.08
Maluvankaranai cheri	29.12	50.00	50.00	56.33	30.77	38.46	49.79	20.00	20.00

Villages in which control measures have been in force

The village and cheri of Maluvankaranai has been added as a comparison for no control methods have as yet been established. It is interesting to note that there has been a striking drop in the incidence of leprosy in two villages namely Polambakkam cheri and Perambakkam village. It is of further interest to note that in the village and cheri of Maluvankaranai the incidence of leprosy has increased more than twice in the former and one and a half times in the latter. What to us is more significant however is the marked decrease in the open case rate in three out of the four villages and the consistent and definite fall in the child rate between the years 1939 and 1945. While no definite conclusions can be drawn we feel that the overall picture

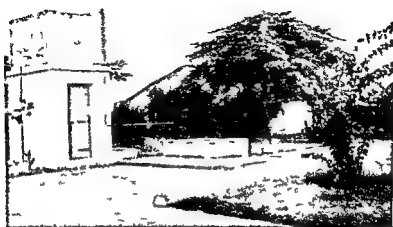


FIG 17—Water tone and compound (1 penner) house



FIG 155—Que t house

shows a downward trend indicating a possible favourable turn in the epidemicity of leprosy in these villages. If this observation proves to be confirmed then the whole rural leprosy programme will have been simplified. We visualise the day when the main rural leprosy prevention units will be linked to sub units with facilities for segregation but visited by a doctor from the main unit each week and this in turn will be connected with the nearest general hospital which ordinarily would be the headquarters of the district so that emergency medical care will at all times be available in the leprosy or general wards of the hospital. It should not be difficult to garage an ambulance at the rural centre in order to provide transport for such cases.

(2) Urban Leprosy

Owing to the overcrowded nature of most of the towns of the East the difficulty of securing a site for local segregation and the impossibility of enforcing this because a co-operative spirit is hard to foster under urban conditions the control of urban leprosy therefore presents many problems. The question of segregation for limited areas in a town has also to be ruled out because there is seldom sufficient land to organise this in the same way as in rural leprosy. In building up a scheme for urban leprosy control the first step which should be taken is to gather information of the relative prevalence of leprosy. There are two methods by which this can be done namely examination of school children for signs of leprosy and/or scrutinising the admission list of out-patient clinics. In one or other of these ways the areas from where cases of leprosy are discovered can be flagged on a map and those of relative high incidence can thus be seen at a glance. More intensive methods can then be applied either by following up all children who have leprosy to their homes and in this way tracing all the immediate contacts or by undertaking an intensive survey in the areas of relative high incidence as indicated by these preliminary investigations of admissions to out-patient clinics. Both methods are time consuming and therefore if urban leprosy is to be thoroughly investigated an officer in charge of urban control should be appointed who would work under the supervision of the director of leprosy control. Attached to the urban control office should be a survey party whose function would be continually to investigate alleged areas of high incidence in the town. Such a system could only be set up in towns of over 100 000 inhabitants. In district headquarters towns the district leprosy officer with the survey unit would include the smaller urban areas in his whole organisation.

The difficulty of urban control will be fully appreciated by anyone who has attempted completely to survey areas in towns for the better to do families are usually the most obstructive. Where possible therefore the preliminary school survey is better done in co-operation with adequately trained school medical officers at a time when the general examination for other diseases is being conducted. In the city of Madras the organisation has been set in motion and a leprosy officer who is a man of experience has been placed in charge of urban investigation. In so far as it is almost impossible to set an area aside in towns for segregation there are only two alternative courses possible (1) institutional segregation (2) home isolation. The former method is preferable and should be resorted to whenever possible but in so far as the accommodation in the majority of the institutions in the Madras Presidency is at the present time taxed to the uttermost this is seldom possible therefore home isolation should be attempted. The supervision of the carrying out of this should be the responsibility



FIG 159—An open case living in close contact with the members of its family in one of the village. He attended regularly for treatment and night segregation and became sane at the end of two years.



FIG 160—A common type of village well.

of the health department If every open case would take the undermentioned precautions leprosy would in the course of time come under control

- (a) Sleep in a separate room taking special care to keep away from children
- (b) Eating sleeping cooking and toilet necessities should be kept separate
- (c) Personal clothing bedclothes towels etc should be soaked in anti septic solution before washing or washed apart from the family's clothes
- (d) The patient should have his own chair or mat and should not allow himself under any circumstances to come into contact with children



FIG 161 —Same well renovated and made a protected water supply Village munshi (headman) standing by well
The help and co operation of officials should always be secured

It is recognised that many of these precautions are in the nature of counsels of perfection and could not be completely carried out but on the other hand if every doctor when called in to treat an open case of leprosy insisted on his absolute separation from children precautions could be taken in a great many houses which would reduce the chances of infection to a minimum Private practitioners tend to be altogether too lax about this matter

While all available evidence goes to show that the chief method of infection is close contact in the house yet it is not advisable that open cases should be



FIG 16 —The site of this well was previously a manure heap

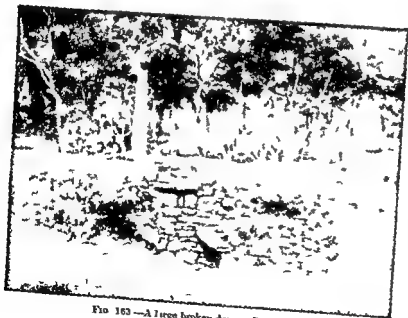


FIG 163 —A large broken down village well



permitted to continue in employment and should be excluded from the following occupations

- (1) School teacher student ayah child nurse or personal servant
- (2) Dhobi barber tailor (laundry or pinman)
- (3) Food vendor mill distributor or public servant
- (4) Driver of any public conveyance either motor propelled horse drawn or man drawn
- (5) Any employment involving contact with children



FIG 164—The same well built up for irrigation purposes (see Fig 163)

The amended Public Health Act makes provision for the prohibition of the above employment

With regard to institutional segregation in the post war reconstruction scheme for leprosy in the Madras Presidency provision has been made for the building of institutions for the larger towns and it is hoped that as authorities gradually realise the need for these and the impossibility of controlling urban leprosy without such arrangements that they will implement this proposal as early as possible. The first institution organised for this purpose should be in connection with Madras city itself. Institutions then should follow in Madura Tuticorin and in all the bigger towns of the Presidency. This manifestly will involve a great expenditure of money but if



FIG 16. —A view of vegetation resulting from the fire in the village area.



FIG 16. —Fire in village rubble pit area.

authorities are in earnest in this matter of urban control the sooner it is realised that comprehensive measures alone will meet the problem the quicker will leprosy in the larger towns come under control and the more rapidly will be dispelled the prevailing notion that leprosy can be controlled by such superficial methods as out patient centres without arrangements for segregation Too often have we lived in a fool's paradise as far as leprosy is concerned and it behoves city health officers to take leprosy as seriously as they would take malaria plague cholera or any other of the major endemic diseases for then and then alone will there be any hope of controlling urban leprosy Further in towns as in villages leprosy is not mainly confined to the poor but can be found in all walks of life from the meanest hovel to the stately mansion

(3) Child Leprosy

It will be realised by the reader that very great emphasis has been laid on leprosy in children and that we maintain the thesis that if leprosy is brought under control as far as children are concerned then the number of adults who become infected are so few that the epidemic is unlikely to be maintained by adult infection alone In the Madras Presidency the following children's work has been organised

- (i) Silver Jubilee Clinic for the study of child leprosy
- (ii) Children's Leprosy Sanatorium Ettapur Salem District
- (iii) Children's section in connection with the Lady Willingdon Leprosy Sanatorium
- (iv) Proposed new home for women and children under the Kasturba Gandhi Memorial Fund

(i) Silver Jubilee Clinic for the Study of Child Leprosy

Saidapet is a suburb of Madras some five miles from the centre of the town in which there has been active health work undertaken for many years in connection with maternity and child welfare Owing to the reported high incidence of child leprosy an investigation unit was established in 1936 largely thanks to a donation from the King George V Silver Jubilee Fund and to a handsome gift from that well known highly esteemed philanthropist the late Sir Frank Carter The clinic is chiefly a research unit whose main objects are

- (1) To investigate the causes of the development of leprosy in children
- (2) To investigate the types of leprosy in children and to study the significance of the various types in relation to their clinical importance and the factors which influence the development of lepromatous leprosy in later life

While treatment is given this clinic is primarily an investigation centre and much of the epidemiological data already submitted has been based on the work of this clinic Over 700 children with leprosy have been enrolled close on 400 of which come from the immediate vicinity of Saidapet The children are divided for investigation purposes as follows

- (a) Those who should be examined every six months
- (b) Those who should be examined every three months
- (c) Those who should be examined every month
- (d) Closed cases under treatment
- (e) Open cases



Fig. 107—A field brought by all goes through perma-
r-bb-1 ten feet



Fig. 108—Older h-pit area (compare all top p-t re) 61-1

All children are photographed and as many individual files as possible are maintained which include the following data

- (a) Clinical photograph
- (b) Chart of lesions at varying intervals
- (c) Record of physical examination
- (d) Record of economic condition of family
- (e) Biopsy of typical lesion and if necessary further biopsies

Thus it is hoped to maintain as complete a history as possible of children and their families and so be the better able to understand the nature spread and course of child leprosy



FIG. 16J.—Silver Jubilee Clinic for the study of child leprosy (S.J.C.C.)
Saidapet Madras

For two years the clinic was run from gifts from private friends. In the year 1938 the clinic was taken over by the Madras Provincial Branch of the British Empire Leprosy Relief Association and it is now hoped that as the leprosy campaign becomes more and more the concern of the Government that this unit will come under the direct control of the Director of the Leprosy Campaign and be part of the overall scheme for leprosy in the Presidency.

(ii) *Children's Leprosy Sanatorium, Ettapur, Salem District*

Owing to the importance of child leprosy, the impossibility of isolating children with infective leprosy under any condition in a village or town and the need for continuing their school studies, the only practical method is to organise institutions for children with leprosy. To this end, through the endeavours of an enthusiastic lay worker, a substantial sum of money was raised to build and equip a leprosy sanatorium for children.



Fig 10—S.J.C.C. Central room showing as it room



Fig 11—S.J.C.C. Dormitory

in the Salem District of the Madras Presidency. While this sanatorium has only been functioning a few years and has been seriously handicapped owing to the war it has shown that by this means it should be possible to meet the problem of infective leprosy in children and through such work train the child to be fit for life when he is discharged from the home.

(iii) *Children's Section in the Lady Willingdon Leprosy Sanatorium*

In the year 1932 the authorities in the Lady Willingdon Leprosy Sanatorium realising the need for providing institutional care for children made arrangements to enlarge the institution in order to admit more children. The children's section is

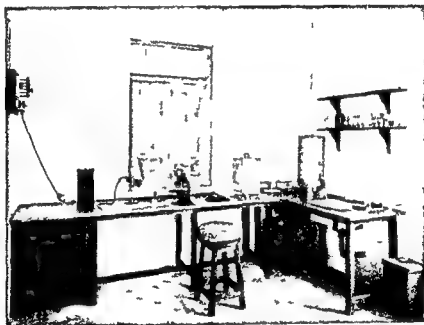


FIG 17 —S J C C Laboratory

entirely separate and facilities are provided for the continuation of their schooling. In addition there are both Scout and Girl Guide companies and opportunities for game. While at school the boys can learn weaving, carpentry or be trained in other ways to be fit for life when they are discharged. No large leprosy institution is complete without providing for the care of children with leprosy, especially that type which is infective or potentially infective. In so far however as only a small proportion of cases pass into lepromatous leprosy in adult life or become deformed, admission to a children's leprosy institution should be strictly selected.

(iv) *Kasturba Gandhi Memorial Home for Women and Children*

A few years ago the wife of Mahatma Gandhi passed away and a fund was raised for her memorial. This was subscribed to all over India and the money collected was placed under a trust deed to be used for work among women and children in country districts. Part of the income of the fund has been set aside for leprosy work among

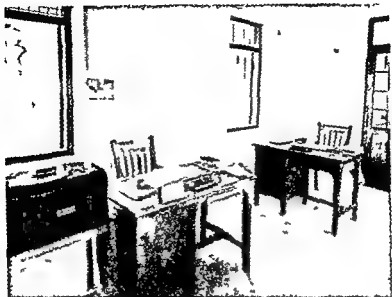


Fig. 13—GJCC Clerk von



Fig. 14—GJCC (D. peno)

women and children in rural areas. An area in the South Arcot district Madras Presidency will be chosen in which the incidence is high and a rural home in that area will be established. The object of the home will be

- (1) To house give treatment and train in some useful employment all women and children (female) who have infective leprosy or potentially infective leprosy
- (2) To admit boys up to the age of seven after which they will be transferred to a larger sanatorium with facilities for caring for older boys

In connection with this home but not dependent on the Hasturba Fund will be developed a rural preventive unit. It would be impossible to control leprosy only by concentrating on women and children and therefore a work such as this would not attain its objective—that of bringing leprosy in women and children under control—unless it were associated with segregation facilities for male cases. The leprosy prevention unit while closely co-operating with the women and children home will be part of the general scheme for the control of leprosy in the South Arcot district and will when established be linked with the district leprosy control office at the headquarters town in Cuddalur where the district leprosy control officer when appointed would establish his quarters. Thus through the opportunity afforded by this fund, it will be easier to set in motion a preventive leprosy scheme for the district of South Arcot with its sanatorium children home rural and possible urban units as already described and envisaged.

(4) Research

No adequately organised campaign can hope to develop and succeed in its objective without facilities for research. Considering the nature of the disease its widespread ravages throughout the world and its antiquity it is deplorable that the opportunities offered to medical men and the facilities for research are so meagre. In the whole of India there is only one recognised centre of research and that is the School of Tropical Medicine in Calcutta. Research work is being undertaken elsewhere particularly in Madras but it is carried out as a part time occupation under very great strain and difficulty. It is only within the last year that a beginning has been made to consider the question of a full time Government research officer. If the same number of well trained personnel had been available for leprosy research in India as for malarial research much more progress would have been made. It is hoped that this contribution while not detailing a research programme will at least convince readers that it is time that research scholarships were made available for leprosy and that a team or teams of research workers with a well trained pathologist biochemist and a clinician must be enlisted if further progress is to be made in the understanding of this most baffling of all diseases.

Apart from research into the pathological immunological and therapeutic aspects of the disease there is wide scope for clinical research along the lines of neurology and orthopaedics and the opportunities for epidemiological research through field units is unlimited. Leprosy research should preferably be undertaken in connection with a medical college and linked up with institutions where the most up to date equipment and staff are available. With all the goodwill in the world it is impossible to develop effective investigation work apart from proper facilities. While many useful and

valuable observations can be made by the enthusiastic worker no substantial progress will be achieved unless leprosy research is put on a sound basis to develop along with the major researches of other diseases and health problems of the country. The Director of Leprosy Research should be a full time worker but of sufficient research experience and leprosy training to organise a comprehensive research programme covering laboratory field clinical immunological and animal investigations and thus only will leprosy research find its rightful place and attract research workers of real eminence. Leprosy research workers should be remunerated on the same scale as any other research officer. The Indian Research Fund Association has subsidised leprosy research in India for the past twenty five years.

(5) Teaching

The general programme of teaching has already been discussed. At the present time in the Madras Presidency facilities for teaching are organised in the medical college hospitals but the lack of trained personnel is a serious handicap to effective teaching. It is hoped that as soon as trained men are available with higher qualifications then there will be lecturers organising their own leprosy departments in every teaching hospital in the Presidency. It would be a tragedy of the greatest magnitude in the organisation of the new medical colleges in India if leprosy were given the same casual attention it has up to now received. Not until the medical student himself is taught by enthusiastic and experienced teachers will there be any hope of finding recruits from the newly qualified graduates who are willing to specialise in leprosy and yet the leprologist is as essential to the community in countries such as India as is the malinologist.

(6) The Relationship of Existing and New Institutions to the General Leprosy Campaign

The various types of institutions necessary for the efficient organisation of the leprosy campaign have already been indicated. The general policy with regard to these should be in conformity with the overall plan of leprosy control. It is therefore a prime necessity that the institutional policy should be such that it will make the greatest possible contribution towards the control of leprosy. If this is to be so strict selection of the type of cases to be admitted into institutions should be made and it is recommended that the following categories should receive preferential admission.

- (1) Early lepromatous cases (infective) who are liable to pass on to the more advanced stages unless given institutional treatment.
- (2) Infectious cases whether early or late in whose house there are young children liable to be infected.
- (3) Acute conditions needing hospitalisation should be admitted temporarily if there is a bed available in the hospital.
- (4) Certain cases which need special investigation and study.
- (5) All active cases among children whether open or closed needing institutional treatment.

All persons who have advanced leprosy should be looked after in homes for the crippled and advanced cases the one can be attached to an existing institution but in many ways it is preferable to organise special homes for such cases and those that become

advanced and crippled should be transferred from sanatoria to these homes. In this connection while it is understandable that official bodies should pay capitation grants to voluntary institutions for the cure of infective cases the withholding of grants because a person is no longer infective is not justified. The State must assume responsibility for the care of the deformed and crippled and if a person is unable to earn a livelihood through the crippling nature of his disease although it is no longer infective he should be supported by the State. The Madras Government extends its capitation grant to all those considered by the medical authority of the institution to have ulcerations which are dangerous to life or limb and are unable to be cared for under local conditions.

(7) The Place of the General Hospital and Out-patient Clinic

Although it has been stated that out patient centres treating a few dozen or several hundreds of cases and unconnected with definite preventive projects can contribute little or nothing towards the control of leprosy yet the principle remains that for persons suffering from chronic diseases such as tuberculosis leprosy or syphilis treatment should be available. Therefore facilities for the treatment of leprosy should be organised at every headquarters hospital District Board or Local Fund Dispensary. If thought advisable a special shed might be set apart for this purpose in other words the routine treatment of leprosy like the treatment of any other chronic disease should not be dealt with only by a special department although such departments are necessary. Routine work can be done at the ordinary hospital or dispensary thus leaving investigation and experiment to the specialist. Not until doctors in ordinary Government hospitals and dispensaries are willing to admit those suffering from leprosy on this basis will any substantial progress be made. Similarly at every hospital situated in districts where leprosy is prevalent facilities should be available for the treatment of persons needing immediate medical or surgical attention even though they may suffer from leprosy. Tuberculosis typhoid and syphilis are continually being treated in the wards of the ordinary hospital and therefore there is no justification apart from traditional prejudice for placing leprosy on a different footing especially if the medical profession could only forget their prejudice and remember that leprosy is probably less dangerous than these other diseases. It is time leprosy was treated as an ordinary disease and not placed in a special category by itself. If authorities were prepared to admit persons with leprosy who were also suffering from acute infections into the infectious diseases wards of the local hospital then rural centres could be much more extensively organised for they would then not be solely dependent on special leprosy institutions when those in the segregation unit needed immediate medical or surgical relief.

(8) Propaganda

It will be realised that if the recent advances in methods of diagnosis and prevention of leprosy are to be made available the question of propaganda is of prime importance. Too often modern teaching including leprosy propaganda is based on the traditional conception of the disease and the reader is never altogether freed from the ancient Biblical ideas that those suffering from leprosy are unclean. Unclean unclean without the camp shall be his habitation. Again I do not wish to be misunderstood but I

would remind the reader that the lay conceptions of leprosy are not due to any error contained in the Scriptures but to the fact as previously pointed out that in Old Testament times the word *Zarath* in Hebrew used as a generic name for a group of diseases was translated leprosy and persons were declared ceremoniously unclean when they had any malformation or any disease which was likely to be dangerous or produce a permanent blemish. It must be remembered that there is the grim and terrifying picture of advanced leprosy which should form the sombre background of propaganda and yet let us not forget that horror and dread are not the prerogative of leprosy alone. One only needs to consider such diseases as cancer, filariasis, advanced sleeping sickness, infantile paralysis and many other deforming diseases to appreciate this statement yet the appalling end result of some of these diseases is not overemphasised to the same extent as that of leprosy. Philanthropic societies may think that without this grim picture ever held before the public the appeal will lose its effect but surely is an unworthy motive for propaganda. The challenge is the challenge of a crusader who enters the battle so that the war may be won. He does not except under special circumstances dwell on the horror of the war but on the glory of it. He does not emphasise the casualties in dead, wounded and blind but on the victories won and on the routing of the enemy. Let us raise the whole subject of propaganda in leprosy to the level of a worthy crusade, a work not only for those who are called to care for the wounded and dying but a call to the knight errant who has the opportunity of finding in leprosy great victories for medical research and then placed in its right perspective leprosy will appear a worthy disease with which to battle and not be associated in the minds of men with a special form of an incurable, unalterable and altogether hopeless. The appeal to help the casualties in the battle must ever be there but it is the battle and not the wounded the glory and not the death which appeals, thus all propaganda must be informative, instructive, comprehensive and attractive to all. In all booklets, lantern lectures and talks, the child must come first. There must be presented a reasoned account of the disease, emphasising its comparative benignity and its tendency to spontaneous cure. An understanding knowledge of the disease must be imparted so that persons do not go about in a cold sweat of fear at the very mention of the name of leprosy but yet are willing to take reasonable precautions against infection. The whole technique of propaganda needs completely overhauling and at the present time a completely fresh approach to the subject of leprosy from the propaganda point of view is being presented by the Medical Branch of the British Empire Leprosy Relief Association and it is hoped that these publications through the Honorary Publicity Secretaries will revolutionise the whole subject and help to convince the public that it is no more shameful to acquire leprosy than to acquire measles. Propaganda must keep a right perspective with regard to treatment for too often is this emphasised to the exclusion of prevention and fosters the idea all too readily accepted that all that is needed in leprosy is a doctor with a bottle of hydnocarpus oil and sufficient dexterity to inject the remedy without pain.

A fundamental principle of all propaganda must be concentration on certain main points for the public will only be confused by too much detail. In addition while a certain amount of press license is permissible propaganda based on exaggerated claims is bound to fail as the public will ultimately become discouraged and disappointed when they realise that it is impossible to bring leprosy under control by establish-

treatment centres only In all propaganda the following points should be emphasised first

- (1) Leprosy is an ordinary disease communicated by close contact by one person on in the infective stage It is neither venereal in origin nor is it a particular manifestation of divine displeasure
- (2) All leprosy is not equally serious Therefore all persons who suspect themselves to be suffering from leprosy should seek specialist advice to ascertain just what measures and precautions should be taken
- (3) Children are particularly susceptible to the disease whereas adults are largely non susceptible If every mother in India could be made to realise that it was as dangerous to allow a child to come into contact with a person suffering from infective leprosy as to permit him to play with a poisonous snake a great step forward towards the control of leprosy would have been taken
- (4) Leprosy except among children is not a highly infective disease so that much of the fear and prejudice it arouses is entirely unwarranted
- (5) If a responsible medical practitioner has declared a person non infective then no discriminatory action should be taken against him either by refusing him entrance into a school or prohibiting his employment

The present series of propaganda slides tend to emphasise the results of treatment to the exclusion of the more important aspect of prevention Before and after treatment slides should not be shown unless carefully explained for an unduly optimistic impression is likely to be created in the minds of the public

A new approach to publicity is being developed but the proper technique will take years to perfect It is certain that without the preparation of the right type of material to be used in the schools colleges for public lectures and private distribution the most scientifically planned campaign will completely fail for without adequate support from the ordinary citizen and a well informed public co operation is impossible and the conquest of leprosy will remain an unattainable ideal

(9) The Place of Voluntary Work in the Leprosy Campaign

It must ever be remembered that if it had not been for voluntary endeavour the progress made in the last twenty years would have been impossible The writer owes his interest in leprosy in the first instance to the Mission to Lepers for this and other philanthropic organisations mainly inspired by the example of Christ were the pioneers in leprosy work The names of Father Damien Mary Reed and many unknown but nevertheless faithful leprosy workers should be honoured along with the great souls of this world for it was through their sacrifice that modern leprosy work was made possible It therefore behoves all official bodies to take full cognisance of voluntary work and encourage missionary societies and other organisations to undertake leprosy work The voluntary worker needs encouragement and leadership but until recently this has not been forthcoming and he seldom has the time or the necessary training to undertake the direction of a widespread campaign If however this is well organised by a full time official in complete sympathy with the work of philanthropic societies then it will be found that the institutions and homes of the Mission to Lepers and other societies will play a part of incalculable value in the leprosy campaign for without the full co operation of the authorities of these institutions the overall plan would be greatly handicapped

Practical Application

We are now in a position to outline the plan of campaign which will eventually we hope be devised for the Madras Presidency. In doing this we trust that it will be helpful to preventive workers in other countries. The direction of the campaign will be in the hands of the Director of Leprosy Campaign and he would be solely responsible to the Medical Administrative Head of the Presidency. At the headquarters of the campaign a survey unit will be available which on receipt of information or on the initiative of the Director will be available to investigate alleged high incidences of leprosy anywhere in the Province. In each district where leprosy is prevalent there will be a district leprosy control officer whose task will be to develop a leprosy unit attached to the Government headquarters hospital. This unit will consist of

- (1) Wards with operation theatre attached to deal with emergency cases sent from outside & from rural prevention units or outlying clinics
- (2) A treatment centre
- (3) A laboratory
- (4) A district campaign office

Attached to the headquarters unit will also be a survey party. The duty of the officer in charge would be to investigate leprosy in the surrounding district and recommend the organisation of suitable preventive units in areas where leprosy is a serious endemic disease.

The rural preventive units with their sub-centres would keep in direct touch with the District Leprosy Control Office.

In each district where leprosy is prevalent would be organised a sanatorium with if necessary its auxiliary units of Children's Sanatorium and Cripples Home. This sanatorium would ordinarily admit cases from areas where there are no control units or from towns which are not big enough to have their own leprosy hospital.

Throughout the larger towns e.g. Madras, Madurai etc. special leprosy sanatoria would be organised to deal with infective cases who will not or cannot isolate themselves. In this way areas of high incidence in the villages will have their own systems of prevention through the rural unit and infective cases from the smaller towns and areas of less importance will be persuaded to go to the nearest leprosy sanatorium.

Along with this teaching in medical college hospitals with efficiently equipped departments will be organised. A research centre in connection with a central leprosy sanatorium and preferably linked to a medical college is contemplated and a children's unit for the segregation and study of child leprosy will be established.

The propaganda side will be directed by the Director of Leprosy Campaign but will largely be undertaken by the British Empire Leprosy Relief Association (Madras Branch). It is hoped that a central propaganda office will be established so that all information concerning the warfare against leprosy can be tabulated, the programme of the campaign fitted on a map and facilities for the training of the welfare officers to be attached to the various special units and leprosy hospital be explained to all prospective candidates. Thus every aspect of leprosy will be cared for and the enemy attacked in every quarter. Such a programme will cost much in money, time and energy but as demonstrated by the work of the Silver Jubilee Children's Clinic at Saidapet and the Rural Unit in Chingleput District the leprosy campaign is a most potent lever by which to raise the health level of the entire community and therefore brings dividends in health, happiness and cleanliness far beyond its own limited sphere.

To day, when so much of value has been destroyed in the cataclysm of war there rises a new hope a fresh challenge. For the call goes forth to all worthy men to attack and break down the seemingly impregnable fortress of disease by a new path through the avenues opened up by modern leprosy research. Thus those who are working in a branch of medicine which was but a short time ago neglected and almost despised see new vistas of opportunity and find themselves placed in the very vanguard of the forces pledged to build a new order where the evil triad of disease malnourishment and economic servitude will be for ever banished.



APPENDIX I

ABSTRACT OF LEPROSY SECTION OF THE MADRAS PUBLIC HEALTH ACT (AMENDED 1944)

ACT No IV OF 1944

PART IV LEPROSY

DEFINITIONS

In this part—

(a) Leprosy means open leprosy that is to say that form of the disease in which leprosy bacilli can be demonstrated from the mucous membrane of the patient's nose or from his skin by any recognised standard method of examination approved by the Surgeon General with the Government

(b) Authorised practitioner means a medical practitioner registered under the Madras Medical Registration Act 1914 and authorised by the Government in this behalf

GENERAL

A local authority may and if so required by the Government shall make such arrangements in its local area as may be directed by the Government for

- (a) the free diagnosis and treatment of persons suffering or suspected to suffer from leprosy and
- (b) the prevention of infection from leprosy

(1) No person who knows that he is suffering from leprosy shall until he is certified by an authorised practitioner to be non infectious and to have been so for a period of not less than three months engage himself or accept any employment

- (i) as a cook attendant workman salesman server or carrier in any place where food is sold to members of the public or is prepared or stored for such sale
- (ii) as a dairy worker tending on or milking cows whose milk is to be sold or distributed to members of the public or as a seller or distributor of milk to members of the public
- (iii) as a driver or conductor of a public conveyance
- (iv) as a public servant or
- (v) in any other capacity which in the opinion of the Health Officer involves deleterious contact with other persons not suffering from leprosy

(2) No person who knows that he is suffering from leprosy shall until he is certified by an authorised practitioner to be non infectious and to have been so for a period of not less than six months

- (a) attend any school college playground or other similar place either as a teacher or as a member of the staff employed therein or

To day, when so much of value has been destroyed in the cataclysm of war there rises a new hope a fresh challenge : For the call goes forth to all worthy men to attack and break down the seemingly impregnable fortress of disease by a new path through the avenues opened up by modern leprosy research. Thus those who are working in a branch of medicine which was but a short time ago neglected and almost despised see new vistas of opportunity and find themselves placed in the very vanguard of the forces pledged to build a new order where the evil triad of disease malnourishment and economic servitude will be for ever banished



(b) engage himself or accept any employment

- (i) as a doctor nurse midwife ayah or orderly
- (ii) as a barber or hairdresser
- (iii) as a tailor
- (iv) as a dhobi or launderer,
- (v) as a house servant personal attender or a peon or
- (vi) in any other capacity which in the opinion of the Health Officer involves deleterious contact with children

(3) No person who has the care of any person whom he knows to be suffering from leprosy shall cause or permit such person to engage himself or accept any employment in any of the capacities referred to in sub section (1) or to attend any place or engage himself or accept any employment in any of the capacities referred to in sub section (2) until he is certified by an authorised practitioner as set forth in sub section (1) or sub section (2) as the case may be

(4) No one shall engage or employ any person whom he knows to be suffering from leprosy in any of the capacities referred to in sub sections (1) and (2) until he is certified by an authorised practitioner as set forth in sub section (1) or sub section (2) as the case may be

SPECIAL AREAS

The Government may by notification declare any area in the Province to be a special area for leprosy and thereupon the following provisions shall apply to such area

(1) No person who knows that he is suffering from leprosy shall

- (a) enter any public conveyance used for the conveyance of passengers at separate fares or
- (b) enter any other public conveyance without previously intimating to the owner driver or conductor thereof that he is suffering from leprosy

(2) No person who has the care of a person whom he knows to be suffering from leprosy shall permit him to be carried

- (a) in any public conveyance used for the conveyance of passengers at separate fares or
- (b) in any other public conveyance without previously intimating to the owner driver or conductor thereof that he is suffering from leprosy

(3) The owner driver or conductor of a public conveyance used for the conveyance of passengers at separate fares shall not convey therein a person whom he knows to be suffering from leprosy at any time when a passenger not suffering from leprosy is being conveyed therein

Provided that a person suffering from leprosy may be conveyed in the public conveyance aforesaid in such cases of emergency and subject to such restrictions and safeguards as may be notified by the Government

(4) The owner or a driver of any public conveyance may refuse to convey any person suffering from leprosy until he has been paid a sum sufficient to cover any loss and expense which will be incurred by reason of the provisions of the next succeeding sub section

(5) The person in charge of a public conveyance in which a person whom he knows to be suffering from leprosy has been conveyed shall as soon as practicable give notice to the Health Officer of the local area in which the conveyance is usually kept and before permitting any other person to enter the conveyance shall cause it to be disinfected.

(6) The local authority when so requested by the person in charge of a public conveyance in which a person suffering from leprosy has been conveyed shall provide for its disinfection.

(1) No person who knows that he is suffering from leprosy shall

(a) attend any school college playground or such other place or

(b) take any book or cause any book to be taken for his use or use any book taken from any public library or circulating library.

(2) No person who has the care of a person whom he knows to be suffering from leprosy shall permit him to do any of the acts prohibited by sub-section (1).

SEGREGATION

The Government may on the recommendation of the Director of Public Health by notification declare any area in the Province to be a segregation area if they are satisfied that in such area adequate segregation accommodation for persons suffering from leprosy has been provided by the local authority or has been placed at its disposal and set apart by it for the purpose and thereupon the following provisions shall apply to such area:

- (i) The Health Officer may by notice require any person suffering from leprosy and residing within the segregation area to remove himself to such segregation accommodation as may be specified in the notice and remain there until such time as he is certified by an authorised practitioner to be no longer infectious.
- (ii) The notice shall allow a reasonable period for compliance therewith.
- (iii) If the person suffering from leprosy does not comply with the notice within the period allowed therein the Health Officer may have him compulsorily removed to the segregation accommodation specified therein using such force as may be reasonably necessary for the purpose.
- (iv) The Health Officer may permit any person detained in the segregation accommodation to engage himself or accept employment in any of the capacities other than those already specified provided that it does not involve the performance of any act specifically prohibited by this Act.
- (v) The notice referred to in clause (i) may be given to the person who has the care of a person suffering from leprosy and thereupon it shall be the duty of the former to remove the person suffering from leprosy to the segregation accommodation specified in the notice.
- (vi) If any person suffering from leprosy escapes from or leaves the segregation accommodation provided for him without the written permission of the Health Officer or any other officer authorised by him in this behalf such person may be arrested without a warrant by any police officer or by any one specially empowered by the Government and removed forthwith to such segregation accommodation.

- (vii) The local authority shall arrange for the food clothing and other necessities of every person suffering from leprosy who is detained in the segregation accommodation but any such person shall be at liberty to make his own arrangements for his food clothing or other necessities
- (viii) If any person is arrested under clause (vi) after having been arrested and dealt with under that clause on at least three previous occasions he shall if the Health Officer so directs in writing be produced before a Presidency Magistrate or a Magistrate of the First Class who shall have power to order his detention in a leprosy annexe attached to a prison until such time as he is certified by an authorised practitioner to be no longer infectious and thereupon all the provisions of the law for the time being in force shall so far as may be and with such modifications if any as may be prescribed apply to such person as if he had been sentenced to simple imprisonment for the period for which such detention was ordered

If the Magistrate does not order such detention or if the order of detention passed by him is subsequently cancelled whether by himself or by any other Presidency Magistrate or Magistrate of the First Class the person arrested or detained as the case may be shall forthwith be removed to the segregation accommodation aforesaid

REVOCATION OF CERTIFICATES

Where any authority prescribed in this behalf has reason to believe that a certificate issued in respect of any person suffering from leprosy has ceased to be correct by reason of his having subsequently become infectious such authority may require such person to obtain a fresh certificate from any authorised practitioner of his choice as to the character of his leprosy that is to say as to whether he is or is not infectious

Unless a fresh certificate as aforesaid is obtained by the person suffering from leprosy within such a period as may be prescribed and such certificate declares him non infectious the certificate previously issued shall for the purposes of all the provisions contained in this part be deemed to have been cancelled

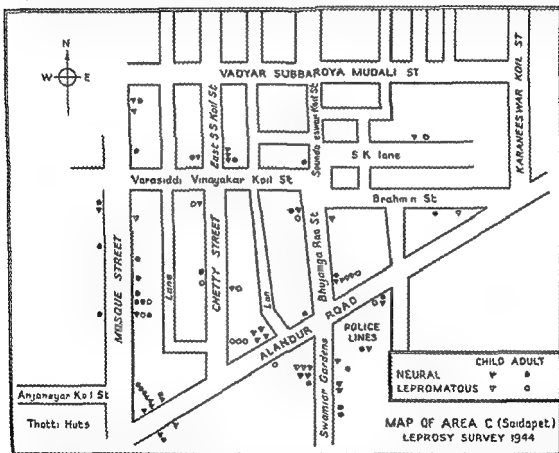
APPENDIX II

SAMPLE SURVEYS

I LI PROSY SURVEY OF AREA C SAIDAPET 1944

Situation

This is a part of the Saidapet town situated about six miles south of Fort St. George on the main trunk road to the south. This suburb of Madras is a town of a population



of about 40,000. Though the municipal limits are extensive, the town proper is about one and a half miles by one mile and this is the most thickly populated area. The population in this part of the Saidapet Municipality is roughly estimated to be about 10,000. This area for purposes of leprosy survey was divided into five smaller areas and called Area A, B, C, D and Ienpet and Edipallayam area. This report

is on Area C bounded on the north by Vadyar Subbaroya Mudali Street on the east by a part of Karneswarar Koil Street on the south by Alandoor Road and on the west by Mosque Street. The population is of a mixed type there are a few weaver families some dhobi families and a fairly good number of them are employed in offices in the city and other places.

Main Occupation of the Population

There is no particularly predominant occupation. A few weavers dhobies and others employed in offices.

Water supply and Drainage

Since the inception of protected water supply few of the wells in this area are sources of drinking water. Well water is used mainly for washing purposes. Drainage in common with other areas is mainly by cesspool collections and removal by carts.

Diet

Staple diet is parboiled rice fish and mutton vegetables varying in quality and quantity according to the economic status of the families.

Condition of Living

Unlike other areas of Saidapet there are a larger number of well to do families in this area. Except in Alandoor Road and Mosque Street the number of families living in a house is rarely more than one or two. Consequently there is less overcrowding in this area.

Secondly there is not that degree of free movement of the population amongst themselves as was found in Area A and B. This is a favourable feature in this area for it gives less chance for leprosy to spread.

State of Literacy and General Comment

Except in Alandoor Road and a few families in Mosque Street most of the people appear to be literate. Many of them are employed and therefore on the whole in this area it appears that the condition of life is much better than in the other areas of Saidapet.

State of Public Health

Filariasis scabies and skin diseases were frequently met with. In Alandoor Road and Mosque Street there appears to be a larger prevalence of hookworm and round worm infections.

Leprosy Incidence

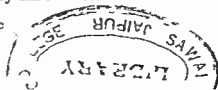
There is a tendency in the better class families to keep secret the prevalence of leprosy in their families while in the poorer classes there is little fear of leprosy and therefore freer movement.

History of Leprosy in the Area

As usual with the other areas this being an urban area no history of the disease can be ascertained. Contact history can to a certain extent be satisfactorily traced.

Silver Jubilee Children's Clinic

Dated 15th June 1945



LIST OF CASES IN AREA C

DEFINITE CASES

Serial No	Door No	Name	Sex	Age	Remarks
<i>Alandoor Road</i>					
1	57	Varadan	Mc	10	Tbd patch on rt back
2	58	Elumalai	Mc	14	Nl Nt
3		Javaraman	Mc	13	Nl Nt
4		Varadarajan	Mc	11	Nl Nt
5	60	Elumalai	Mc	10	Nl Nt
6	15	Selvaraj	Mc	10	Nl Nt
7	Hut	Muniammal	Fe	7	Retgressed Minor Tbd macule on rt buttock
8	Hut	Syed Iyed Basha	Mc	14	Nl Nt
<i>Samior Thottam</i>					
9	Hut	Mariamammal	Fe	8	Nl Nt Major
10		Sakuntala	Fe	2	Nl Nt
11		Arumugham	Mc	14	Ll
12		Annammal	Fe	15	Ll
13		Palavam	Mc	8	Nl Nt
14		Geniamuthu	Mc	10	Nl Nt
15		Munuswamy	Mc	8	Nl Nt Major
16		Kanniappan	Mc	12	Nl Nt
<i>Methodist Mission School</i>					
17		Komala	Fe	6	Nl Nt (patch on rt buttock lateral)
18		Kamala	Fe	6	Nl Nt (patch on rt forearm back)
19		Jayam	Fe	10	Nl Nt (patch on rt leg lateral back)
20		Jambak Thorai	Fe	13	Nl Nt
<i>Mosque Street</i>					
21	3	Anchalakshi	Fe	11	Nl Nt and Nl Nt (patch on rt knee and rt arm back)
22	4	Susheela Devi	Fe	14	Tbd patches all over the body
23	15	Kanniammal	Fe	10	Nl Nt
24	23	Vajiravelu	Mc	3	Nl Nt
25	54	Vadivelu	Mc	7	Nl Nt
<i>Subramanya swami Koi East Lane</i>					
26	10	Saritha	Fe	5	Nl Nt
27	11	S D Sakuntala	Fe	11	Nl Nt
28	13	Kanniammal	Fe	6	Nl Nt
29	2	Jaya	Fe	10	Nl Nt
<i>Soundreswarar Koi Street</i>					
30	16	Davalu	Fe	10	Nl Nt
<i>Chetty Street</i>					
31	12	Chokkalingam	Mc	14	Nl Incipient
32	38	Kanakamburan	Mc	1	Nl Nt Minor

is on Area C bounded on the north by Vadyar Subbaroy a Mudali Street on the east by a part of Karneswarar Koil Street on the south by Alandoor Road and on the west by Mosque Street. The population is of a mixed type there are a few weaver families some dhobi families and a fairly good number of them are employed in offices in the city and other places.

Main Occupation of the Population

There is no particularly predominant occupation. A few weavers dhobies and others employed in offices.

Water-supply and Drainage

Since the inception of protected water supply few of the wells in this area are sources of drinking water. Well water is used mainly for washing purposes. Drainage in common with other areas is mainly by cesspool collections and removal by carts.

Diet

Staple diet is parboiled rice fish and mutton vegetables varying in quality and quantity according to the economic status of the families.

Condition of Living

Like other areas of Saidapet there are a larger number of well to do families in this area. Except in Alandoor Road and Mosque Street the number of families living in a house is rarely more than one or two. Consequently there is less overcrowding in this area.

Secondly there is not that degree of free movement of the population amongst themselves as was found in Area A and B. This is a favourable feature in this area for it gives less chance for leprosy to spread.

State of Literacy and General Comment

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Leprosy Incidence

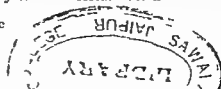
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History of Leprosy in the Area

As usual with the other areas this being an urban area no history of the disease can be ascertained. Contact history can to a certain extent be satisfactorily traced.

Silver Jubilee Children's Clinic

Dated 15th June 1945



LIST OF CASES IN AREA C

DEFINITE CASES

Serial No	Door No	Name	Sex	Age	Remarks
<i>Manoor Road</i>					
1	7	Varadan	Mc	10	Tbd patch on rt back
2	38	Elumalai	Mc	14	N1 Nt
3		Javaraman	Mc	13	N1 Nt
4		Varadarajan	Mc	8	N1 Nt
5	60	Elumalai	Mc	10	N1 Nt
6	15	Selvaraj	Mc	10	N1 Nt
7	Hut	Muniammal	Fe	7	Retgressed Minor Tbd macule on rt buttock
8	Hut	Syed Iyed Basha	Mc	14	N1 Nt
<i>Samior Thottam</i>					
9	Hut	Matiammal	Fe	8	N1 Nt Major
10		Sakuntala	Fe	2	N1 Ns
11		Arumugham	Mc	14	L1
12		Annammal	Fe	15	L1
13		Palaiam	Mc	9	N1 Nt
14		Gengamuthu	Mc	10	N1 Ns
15		Munuswamy	Mc	8	N1 Nt Major
16		Kanniappan	Mc	12	N1 Nt
<i>Methodist Mission School</i>					
17		Komala	Fe	6	N1 Ns (patch on rt buttock lateral)
18		Kamala	Fe	6	N1 Ns (patch on rt forearm back)
19		Jayam	Fe	10	N1 Ns (patch on rt leg lateral back)
20		Jambak Thoraiya	Fe	13	N1 Nt
<i>Mosque Street</i>					
21	3	Anchalakshi	Fe	6	N1 Ns and N1 Nt (patch on rt knee and rt arm back)
22	4	Susheeli Devi	Fe	14	Tbd patches all over the body
23	15	Kanniammal	Fe	10	N1 Ns
24	28	Vajravelu	Mc	3	N1 Ns
25	54	Vadivelu	Mc	7	N1 Ns
<i>Subramania swami Koil East Lane</i>					
26	10	Saratha	Fe	5	N1 Ns
27	11	S D Sakuntala	Fe	11	N1 Ns
28	13	Kanniammal	Fe	6	N1 Nt
29	2	Jaya	Fe	10	N1 Ns
<i>Soundreswarar Koil Street</i>					
30	16	Devaki	Fe	10	N1 Nt
<i>Chetty Street</i>					
31	12	Chokkalingam	Mc	14	N1 Incompet
32	39	Kanakaambiran	Mc	1	N1 Nt Minor

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Diet

polished parboiled rice fish and mutton vegetables varying in quality and quantity according to the economic status of the families.

Condition of Living

Like other areas of Saidapet there are a larger number of well to do families in the Alandoor Road and Mosque Street the number of families in each house is rarely more than one or two. Consequently there is less overcrowding in the area.

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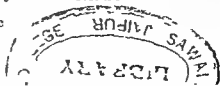
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Silver Jubilee Children's Clinic

Dated 15th June 1945



LIST OF CASES IN AREA C

DEFINITE CASES

Serial No	Door No	Name	Sex	Age	Remarks
<i>Abindoor Road</i>					
1	57	Varadan	Mc	10	Tbd patch on rt back
2	58	Elumalai	Mc	14	Nl Nt
3		Jataraman	Mc	13	Nl Nt
4		Varadarajan	Mc	8	Nl Nt
5	60	Elumalai	Mc	10	Nl Nt
6	15	Selvaraj	Mc	10	Nl Nt
7	Hut	Muniammal	Fc	7	Petrogre sed Minor Tbd macule on rt buttock
8	Hut	Syed Iyed Basba	Mc	14	Nl Nt
<i>Samsor Thottam</i>					
9	Hut	Marimmal	Fc	8	Nl Nt Major
10		Sakuntala	Fc	4	Nl Ns
11		Arumugham	Mc	14	Ll
12		Annammal	Fc	15	Ll
13		Palayam	Mc	8	Nl Nt
14		Gengamuthu	Mc	10	Nl Ns
15		Munuswamy	Mc	8	Nl Nt Major
16		Kannappan	Mc	12	Nl Nt
<i>Vithodis Mission School</i>					
17		Kamala	Fc	6	Nl Ns (patch on rt buttock lateral)
18		Kamala	Fc	6	Nl Ns (patch on rt forearm back)
19		Javam	Fc	10	Nl Ns (patch on rt leg lateral back)
20		Jambak Thorana	Fc	13	Nl Nt
<i>Mosque Street</i>					
21	3	Anchalikshi	Fc	6	Nl Ns and Nl Nt (patch on rt knee and rt arm back)
22	4	Susheela Devi	Fc	14	Tbd patches all over the body
23	15	Kanniammal	Fc	10	Nl Ns
24	26	Vajravelu	Mc	3	Nl Ns
25	54	Vadivelu	Mc	7	Nl Ns
<i>Subramania swami Koi East Lane</i>					
26	10	Saratha	Fc	5	Nl Ns
27	11	S D Sakuntala	Fc	11	Nl Ns
28	13	Kanniammal	Fc	11	Nl Nt
29	2	Jaya	Fc	10	Nl Ns
<i>Soundreswarar Koi Street</i>					
30	16	Devaki	Fc	10	Nl Nt
<i>Chetty Street</i>					
31	12	Chokkalingam	Mc	14	Nl Incipient
32	38	Kanakamburan	Mc	1	Nl Nt Minor

PRACTICAL TEXTBOOK OF LEPROSY

DEFINITE CASES—continued

Serial No	Door No	Name	Sex	Age	Remarks
<i>Bhujanga Rao Street</i>					
33	1	Narasimham	Mc	9	N1 Ns
34	8	Dilli	Mc	14	L1
35		Elumalai	Mc	12	L1
36		Govindammal	Fc	8	N1 Incipient
37		Kannan	Mc	9	N1 Incipient
38		Saroja	Fc	7	N1 Nt
<i>Brahmin Street</i>					
39	5	Palani	Mc	9	N1 Ns
40	8	Natarajan (Police Lines)	Mc	14	N1 Nt

DEFINITE CASES—ADULTS

Serial No	Door No	Name	Sex	Age	Remarks
<i>Alandoor Road</i>					
1	58	Babu	M	18	N1 Nt Major (patch on rt elbow back)
2		Arumugham	M	18	N1 Ns
3		Kannia Pillai	M	60	N1 Ns
4	60	Natcsan	M	18	N1 Ns
5	66	Kannappa Natchar	M	30	N1 Ns
6	15	Purushothaman	M	30	N1 Ns
7	17	Krishnaswamy	M	48	Open case
<i>Samior Thottam</i>					
8	Hut	Munuswamy	M	45	N3
9		Gengan	M	45	N3 Ns
10		Gengan	M	35	Big patch on rt shoulder back retrogressed
11		Nagammal	F	22	N3
<i>Alandoor Road</i>					
12	22	Dakshinamoorthy	M	30	L2
13		Vandimodu Saratha	F	30	Tbd (patch on rt elbow back)
<i>Mosque Street</i>					
14	3	Perumal Naidu	M	20	N2 Na (closed case)
15	11	Ranganayaki	F	35	N1 Ns (retrogressed Tbd lesion)
16	20	Kamalammal	F	50	N1 Ns
17	24	Angammal	F	50	N3
18	26	Chinnammal	F	30	N1 Nt Minor
19	27	Murugesan	M	20	Old retrogressed Tbd lesion
20		Kannuammal	F	38	N1 Ns
21		Tirupati	M	36	L1 N3
22	28	Krishnaswamy	M	28	L3
23		Mudichammal	F	50	Tbd Minor (patch on lt shoulder)
24	36	Jankiammal	F	30	N1 Nt (patch on rt elbow back)
25	48	Kadumbadi	F	30	Tbd Minor
26	50	Copal Naidu	M	60	Tbd patch retrogressed

DEFINITE CASES—ADULTS—continued

Serial No	Door No	Name	Sex	Age	Remarks
<i>Subramania aiyam Koil Lane</i>					
27	13	Ailandammal	F	38	Tbd Minor (patch on lt forearm)
28	2	Arjunan	M	17	N1 Ns
<i>Soundrenagar Koil Street</i>					
29	1	Arumugha Mudaliar	M	19	N1 Ns
30	10	Ka tappa Naidu	M	40	L3
<i>Chetty Street</i>					
31	12	Cenadara Ayyar	M	40	L2
32	18	Nagammal	F	30	N1 Ns
33		Kannurippan	M	18	L
34		Kali	M	17	L1
35	27	B S Niler	M	30	L3
36	38	Kamilammal	F	30	N1 Nt
<i>Bhujanga Pao Street</i>					
37	7	Rajagopalan	M	19	N1 Ns
38	20	Ananda Pao	M	37	L
39	21	Ponnammal	F	27	N1 Ns
40	8	Kuppammal	F		L1
<i>Brahmin Street</i>					
41	8	Tyagarajan	M	20	N1 Nt (retrogressed)
<i>Police Lanes</i>					
42	40	Jayaraman	M	38	N1 Ns
<i>Chetty Street—continued</i>					
43	4	Srinivasan	M	21	N1 Ns

SCHEDULE I

District Chinglput

Taluk Sandapet

Village Area C

Total Population	Sex	Cases Detected			Incidence per 1000
		Neural	Lepromatous	Total	
1997	Males	37	13	50	25.06
1392	Females	31	2	33	23.52
3389	Total	68	15	83	

PRACTICAL TEXTBOOK OF LEPROSY

DEFINITE CASES—continued

Serial No	Door No	Name	Sex	Age	Remarks
<i>Bhujanga Rao Street</i>					
33	1	Narasimham	Mc	11	N1 Ns
34	2	Dilli	Mc	14	L1
35		Elumalai	Mc	12	L1
36		Govindammal	Fc	8	N1 Incipient
37		Kannan	Mc	9	N1 Incipient
38		Saroja	Fc	7	N1 Nt
<i>Brahmin Street</i>					
39	5	Palani	Mc	9	N1 Ns
40	8	Natarajan (Police Lines)	Mc	14	N1 Nt

DEFINITE CASES—ADULTS

Serial No	Door No	Name	Sex	Age	Remarks
<i>Alandoor Road</i>					
1	58	Babu	M	18	N1 Nt Major (patch on rt elbow back)
2		Arumugham	M	18	N1 Ns
3		Kannia Pillai	M	60	N1 Ns
4	60	Natesan	M	18	N1 Ns
5	66	Kannappa Nacker	M	30	N1 Ns
6	15	Purushothaman	M	30	N1 Ns
7	17	Krishnaswamy	M	48	Open case
<i>Samior Thottam</i>					
8	Hut	Munuswamy	M	45	N3
9		Gengan	M	45	N3 Ns
10		Gengan	M	30	B ₁ patch on rt shoulder back retrogressed
11		Nagammal	F	22	N3
<i>Alandoor Road</i>					
12	22	Dakshinamoorthy	M	30	L2
13		Vandimodu Saratha	F	30	Tbd (patch on rt elbow back)
<i>Mosque Street</i>					
14	3	Perumal Naidu	M	20	N2 Na (closed case)
15	11	Panganayaki	F	30	N1 Ns (retrogressed Tbd lesion)
16	20	Kamalammal	F	50	N1 Ns
17	24	Angammal	F	50	N3
18	26	Chunnammal	F	30	N1 Nt Minor
19	27	Murugesan	M	20	Old retrogressed Tbd lesion
20		Kannammal	F	38	N1 Ns
21		Tirupati	M	36	L1 N3
22	28	Krishnaswamy	M	28	L3
23		Mudichatammal	F	50	Tbd Minor (patch on lt shoulder)
24	36	Janikammal	F	30	N1 Nt (patch on rt elbow back)
25	48	Kadumbadi	F	30	Tbd Minor
26	50	Gopal Naidu	M	80	Tbd patch retrogressed

DEFINITE CASES—ADULT—continued

Serial No	Door No	Name	Sex	Age	Remarks
<i>Subramania swami Koil Lane</i>					
27	13	Aylandammal	F	38	Tbd Minor (patch on lt forearm)
28	2	Arjunan	M	17	N1 Ns
<i>Soundresuarar Koil Street</i>					
29	1	Arumugha Mudalar	M	19	N1 Ns
30	19	Kustappa Naidu	M	40	L3
<i>Chetty Street</i>					
31	12	Gengadara Asuri	M	40	L2
32	18	Nagammal	F	30	N1 Ns
33		Kanniappan	M	18	L2
34		Kali	M	17	L1
35	27	B S Niler	M	35	L3
36	39	Kamalammal	F	30	N1 Nt
<i>Bhuyanga Rao Street</i>					
37	7	Rajagopalan	M	19	N1 Ns
38	20	Ananda Pao	M	30	L2
39	21	Fonnammal	F	27	N1 Ns
40	8	Kuppammal	F		L1
<i>Brahmin Street</i>					
41	8	Tyagarajan	M	22	N1 Nt (retrogressed)
<i>Police Lines</i>					
42	45	Jayaraman	M	38	N1 Ns
<i>Chetty Street—continued</i>					
43	28	Srinivasan	M	21	N1 Ns

SCHEDULE I

District Chingleput Taluk Sudapet Village Area C

Total Population	Sex	Cases Detected			Incidence per 1000
		Neural	Lepromatous	Total	
1997	Males	37	13	50	30.80
1392	Females	31	2	33	23.50
2689	Total	68	15	83	

ANALYSIS OF CASES BY TYPE AND AGE

Age Group	Cases				Total for Age Group	Percent of Tot Cases
	Neural		Lepromatous			
	No	Percentage	No	Percentage		
0-14	36	90.0	4	10.0	40	48.2
15-34	17	85.0	3	15.0	20	24.1
Over 34	15	65.2	8	34.8	23	27.7
Total	68	80.6	15	19.9	83	100.0

II LEPROSY SURVEY OF AREA B SAIDAPET 1942

Situation

This is a part of the Saidapet town situated about six miles south of Fort St. George on the main trunk road to the south. This suburb of Madras is a town of a population of about 40,000. Though the municipal limits are extensive the town proper is about one and a half miles by one mile and this is the most thickly populated area. Population in this part of the Saidapet Municipality is roughly estimated to be about 10,000. This area for purposes of leprosy survey was divided into five smaller areas and called Area A, B, C, D and Fenpet and Edapallayam area. This report is on the survey of Area B. The population is mainly weavers. The weaving community is named the Senguntha mudahar community.

Main Occupation of the Population

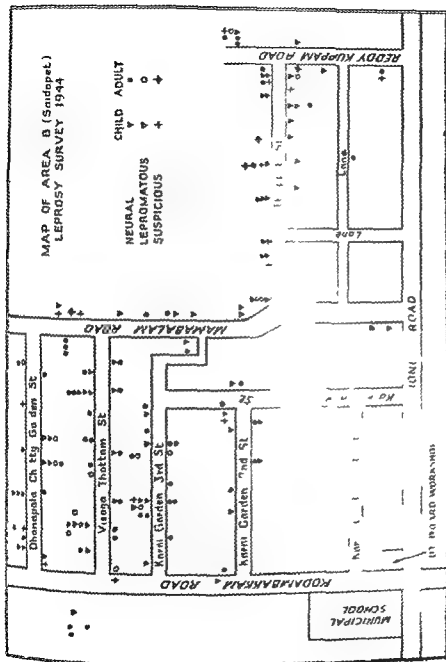
The chief occupation of the population is weaving while dyeing and cloth printing with fanciful designs are also carried on by a few. The chief member in a household is the owner of handlooms employing many others as wage earners under him.

Water supply and Drainage

Drinking water was from wells for years long past and in 1939 protected water supply was introduced by the Municipality. Very few house connections are given so much so the population depends largely on public taps. Each public tap is much crowded during the water supply hours that in and around a public tap filth and dirty water are common sights. The problem of drainage has been the curse of Saidapet. Drain water is collected in cesspools located behind a house where it happens to be a sanitary line or in front of houses in the main streets affording the most contemptible sight to the passer by. The cesspools often overflow. Unless the problem of drainage is tackled Saidapet cannot come anywhere near a fit place for living. Situated as it is so near Madras the health of the city is bound to be affected.

MAP OF AREA B (Saidpet)
LEPROSY SURVEY 1944

NEURAL	CHILD ADULT
LEPROMATOUS	+
SUSPICIOUS	+



by the unhygienic conditions of its suburbs. Several attempts were made to institute underground drainage systems in the municipality and when active measures were begun to tackle this problem the war came with the result that all schemes were shelved compul only for the duration of the war. It is hoped after the war the attempts will be renewed.

Diet

The staple diet is parboiled rice. Ragi khoozhu vegetables mutton and fish these ranging in quality and quantity according to the economic status of families. Most of the families are poor. In a diet survey carried on in this area by the Nutrition Institute Coonoor it was found that the average consumption of diet in calories was lower than normal and ranged from 900 to 2350 calories.

Conditions of Living

The poor economic condition is largely contributing to the fact of several families living in the same house as co tenants. There are a number of families living in the same house varying from three to six or seven. When a survey worker gets into a house it is a deplorable sight to see a number of children and adults moving about in the house and he feels that it is no wonder an open case in their midst is a potent source of infection to many of the children and perhaps to some of the adults.

Further in this area as in Area A the movement of the people from place to place in the course of their weaving work and the shifting of residences is very frequent. Belonging to the same community they mix up freely. The chances of spread of leprosy in these areas are very great even if only a few open cases are present. This is probably the cause of the high incidence in this area as well as in Area A.

State of Literacy and General Comment

The majority of the population is illiterate. That leprosy is infectious few of them know. This is an area from which several child patients come. This has given us occasions to put into the minds of their families the right type of ideas about leprosy. After some years of work some of these families now realise the infectivity of leprosy but it is not strong enough to adopt measures of segregation if the patient concerned is his own close relation. But there is considerable anxiety to take treatment as an alternative to segregation. At any rate though defective in itself from the point of view of prevention yet it appears to be a healthier outlook on leprosy. The co operation of the public for a leprosy survey has been uniformly good.

State of Public Health

Hook worm and round worm infections are very common among children in this area. Scabies and fungus are also largely prevalent. Hygiene both personal and public is little in evidence. Any epidemic such as the chicken pox or smallpox or cholera finds easy and fertile soil in this area when once it starts. Filariasis is very common.

Leprosy Conscience

People recognise leprosy even at early stages. This is evidenced by the fact that 63.72 per cent of the children found to be showing manifestations of leprosy in this

area during the survey were registered even prior to the survey by voluntary presentation. Some of our very early cases are from this area. In that way it is gratifying to note that a better knowledge about leprosy has been gained by the population. We hope this is a first step towards the ideal of segregation of open cases.

History of Leprosy in the Area

No antecedent history of leprosy is possible to trace as this is an urban area with a constantly changing or moving population. Attempts are constantly maintained in this direction. There are several instances of a successful tracing of the contact history in this area several days or months after admission.

Silver Jubilee Children's Clinic

Dated 11th April 1944

LIST OF CASES IN AREA B

CHILDREN

Serial No	Door No	Name	Age	Sex	Remarks	
<i>Karni Gardens—Straight Street</i>						
1	Hut	Lakshmi	10	Fc	Suspicious	634
2	16	Vishalakshi	8	Fc	Nt Nt Minor	233
3	8	Manivelu	10	Mc	Nt Nt Major	294
4		Saraswathy	11	Fc	Suspicious	670
5	19	Thulukannan	12	Mc	Nt Nt	103
<i>Karni Gardens—First Street</i>						
6	13	Angammal	10	Fc	Nt Nt Minor	410
<i>Karni Gardens—Second Street</i>						
7	1	Natarajan	10	Mc	Nt Nt Minor	239
8		Jalini	8	Mc	Nt Nt Minor	42
9		Velayutham	12	Mc	Nt Nt	43
10	82	Harnayeni	9	Fc	Nt Nt	630
11	11	Vaduvambal	13	Fc	Nt Nt (patch on shoulder)	
12	21	Cogan	13	Mc	Patch on lt shoulder	
<i>Karni Gardens—Third Street</i>						
13	Hut	Varadarajan	12	Mc	Nt Nt	246
14	Hut	Sisagami	12	Fc	Nt Nt	32
15		Ponnuthu	14	Mc	Nt Nt	317
16		Thangavelu	6	Mc	Suspicious	3
17		Ikklu	8	Fc	Lt (negative)	74
18	Hut	Kannammal	7	Fc	Nt Nt	913
19	Hut	Panchakaram	8	Mc	Nt Nt Minor	402
<i>Kodumballa High Road</i>						
20	6	Chinnapponna	4	Fc	Nt Nt	229
21	11	Jakiri	7	Fc	Suspicious	37
22	Hut	Iarvathy	12	Fc	Nt Nt	610

CHILDREN—continued

Serial No	Door No	Name	Age	Sex	Remarks	
<i>Dhanopal Chetty Gardens</i>						
23	3	Elumalai	12	Mc	N1 Ns	
24	2	Natarajan	11	Mc	Suspicious	50 A
25	11	Jayaraman	4	Mc	N1 Nt Minor	603
26		Subbammal	1	Fc	N1 Ns	
27	Hut	Somasundaram	14	Mc	L1 (negative)	160
28		Murugesan	11	Mc	N1 Nt Minor	187
29	10	Krishnan	10	Mc	Suspicious	320
30	9	Manicklam	8	Mc	N1 Ns	80
31	6/2	Kannammal	13	Fc	L1	313
32		Baby	11	Fc	N1 Ns	315
33		Kamala	6	Fc	N1 Nt Minor	418
34	5	Rangiah	12	Mc	Suspicious	509
35		Sarojini	9	Fc	N1 Ns	547
36	4	Vaduvambal	4	Fc	N1 Nt (patch on rt forearm)	
37		Saroja	6	Fc	N1 Nt	
<i>Visagathottam</i>						
38	10	Kamakshee	8	Fe	N1 Ns	208
39	5	Parthasarathy	8	Mc	N1 Nt Major	422
40	2	Tirupurem	7	Fe	N1 Ns	
41	46	Elangah	7	Mc	N1 Nt Minor	612
42	43	Manicklam	12	Mc	N1 Nt Major	198
43		Saradhambal	7	Fe	N1 Nt	240 A
44		Murugathammal	10	Fc	N1 Nt	
45	Hut	Sundaram	12	Mc	L1	632
46		Vaduvambal	10	Fc	N1 Ns	528 A
47		Ramachandran	6	Mc	N1 Ns	649
48	Hut	Elumalai	7	Mc	N1 Ns	
49	26	Panchaksaram	14	Mc	N1 Nt Minor	164 A
50		Pachaiammal	10	Fc	N1 Ns	672
<i>Old Mambalam Road</i>						
51	3	Kamala	13	Fc	N1 Nt Minor	483
52	5	Ganesan	8	Mc	N1 Ns	577
53	11	Dhanalakshmi	8	Fc	N1 Ns	3 A
54		Vidyalingam	11	Mc	Typical patch rt abdomen	
55	13	Bangaruswamy	5	Mc	Patch on lt knee more typical than suspicious	
56	17	Logammal	7	Fc	Suspicious patch rt shoulder back	
57	18	Arunagiri	10	Mc	L1 (border line)	379
58		Devakunjari	12	Fc	L1	378
59	21	Saroja	8	Fc	N1 Ns	644
60	48	Varadan	8	Mc	Suspicious	473
61		Parthasarathy	10	Mc	N1 Nt Minor	436
62	56	Chandra	9	Fc	Suspicious	553
63	61	Kamala	6	Fc	N1 Nt Minor	80 A
64	66	Ganesan	6	Mc	N1 Ns	1 A
65	71	Velayutham	12	Mc	N1 Nt Minor	210
66	72	Arumugham	14	Mc	L1	134
67		Devanayakammal	4	Fc	N1 Nt Minor	487

CHILDREN—continued

Serial No	Door No	Name	Age	Sex	Remarks	
<i>Thoppet Street</i>						
69	41	Parvathy	14	Fe	Nl Ns	73 A
69		Annammal	9	Fe	Ll	38 A
70		Savandammal	12	Fe	Ll	49 A
71		Chinnarajalu	8	Me	Patch on rt thigh back	96 A
72	42	Saroja	6	Fe	Patch on rt thigh back	
73	45	Parvathy	5	Fe	Suspicious	67b
74		Kanakasabai	7	Me	Nl Ns	182 A
75	40	Dakshinamoorthy	10	Me	Patch on rt scapula	
76		Sakuntala	7	Fe	Nl Nt Minor	574
77		Shanmugha undaram	12	Me	Nl Ns	146
78	56	Patnavelu	10	Me	Nl Nt Minor	244
79		Balu	9	Me	Nl Nt Minor	247
80		Shunmugham	14	Me	Nl Ns	105
81	60	Angamuthu	14	Me	Nl Ns	218
82		Shanmugasundaram	12	Me	Suspicious	
83	72	Navaneetham	13	Fe	Tbd Minor	177 A
84		Pamalingam	10	Me	Nl Nt	185 A
85	73	Lakshmi	10	Fe	Nl Nt	625
86	74	Tirupuram	5	Fe	Suspicious	
87	76	Pattammal	7	Fe	Nl Nt Minor	236 A
88		Lakshmi	14	Fe	Nl Ns	498
89	2	Kanniappan	9	Me	Nl Ns	
90	5	Pukku	11	Fe	Suspicious	
91	9	Janiki	4	Fe	Nl Nt Minor	217 A
92		Lingam	14	Me	Ll	21 A
93	10	Natarajan	10	Me	Nl Ns	220
94	11	Sivalingam	8	Me	Nl Nt Major	645
95	13	Nayagam	10	Fe	Nl Nt Major	425
96	16/17	Nandagopal	6	Me	Nl Nt	169 A
97		Ianganathan	9	Me	Nl Ns	183 A
98	21	Sambandan	8	Me	Nl Nt Minor	113 A
99		Ramalingam		Me	Patch on lt thigh	
100	33	Flammal	10	Fe	Nl Ns (border line)	642
101	37	Kannammal	13	Fe	Patch on lt forearm	
102	35	Subramaniam	10	Me	Nl Nt Minor	111
<i>Reddikuppam Road</i>						
103	2	Shanmugham	10	Me	Suspicious	204 A
104	5	Venkatesan	6	Me	Tbd Minor lt buttock	
105	9	Subramaniam	4	Me	Patch rt thigh front (suspicious)	
106		Killiammai	11	Fe	Patch on rt leg back	
107	18	Swaminathan	12	Me	Nl Ns	329
			Me	Fe		
Definite cases			49	42	Total	91
Suspicious cases			8	8	Total	16
			57	50		107

LIST OF CASES IN AREA B

Adults

Serial No	Door No	Name	Age	Sex	Remarks
<i>Karni Gardens—Straight Street</i>					
1	15	Lakshmi	35	F	Tbd Major (patch on rt elbow)
2	8	Arumugha Mudaliar	35	M	N1 Ns
3	22	Balasuri	32	M	N1 Ns
4		Balakrishnan	23	M	N1 Ns
<i>Karni Gardens—First Street</i>					
5	3	Thangavelu Naicker	35	M	Tbd Minor
6	13	Gurusami Chetty	40	M	N1 Nt
7		Gengammal	32	F	N1 Ns
<i>Karni Gardens—Second Street</i>					
8	1	Muniammal	31	F	N2
9	8/2	Veerasami Naidu	31	M	N1 Ns
10	14	Rajammal	31	F	N1 Ns (closed case)
<i>Karni Gardens—Third Street</i>					
11	2	Govindarajulu	45	M	N1 Ns
12		Nagammal	40	F	N1 Ns
13	3	Munuswamy Naicker	45	M	L2
14	5	Swarnambal	18	F	N1 Ns
15	7	Ramaswamy Mudaliar	50	M	Tbd Minor
16		Madurai Naicker	59	M	Tbd Minor
17	10	Govindal Mudali	32	M	N1 Ns
18	14	Tiruthavukarasu	18	M	L1 (negative)
19	13	Venkatammal	25	F	N1 Ns (patch on lt elbow and rt leg)
20		Kanniah Naidu	35	M	Tbd Minor
21	18	Kandavelu Mudaliar	48	M	N1 Nt Minor
22	22	Chinnappan	20	M	N1 Nt
<i>Kadambakkam High Road</i>					
23	2	Govindaswamy	40	M	N1 Ns
24	8	Kanniah Maistry	35	M	N1 Ns
25	11	Adiammal	40	F	L1
26	Hut	Balakrishnan	30	M	N2 Ns
27	Hut	Kannammal	45	F	N3
28	Hut	Lakshmi	23	F	N1 Ns
<i>Dhanapal Chetty Gardens</i>					
29	11	Kannappa Mudar	40	M	Minor
30		Subbaraya Chetty			
31		Lakshmanammal			
32	8	Lakshmi			
33	Hut	Arumugham			

ADULTS—continued

Serial No	Door No	Name	Age	Sex	Remarks
<i>Dhanapal Chetty Gardens—continued</i>					
34	13	Subramaniam	30	M	Tbd Minor
35	16	Radha Bai	35	F	L1 (negative)
36	"	Vel Murugappan	30	M	N1 Ns (patch rt hip)
37	"	Doraiswami Naidu	48	M	Tbd Minor
38	4	Subramaniam	30	M	Tbd Minor
<i>Visagathottam</i>					
39	20	Papathy ammal	35	F	Patch on rt hand elbow
40	Hut	Kamakshie ammal	50	F	N1 Ns
41	15	Dinodaram	18	M	Patch rt back side
42	"	Laxathi ammal	25	F	N1 Ns
43	Hut	Iyammal	45	F	N1 Nt (patch on lt back)
44	"	Perumal	20	M	L2 (died)
45	10	Venu Naicker	40	M	N2
46	"	Varadarajulu Naicker	30	M	N1 Ns
47	2	Manikka Mudali	22	M	N1 Ns
48	46	Muruswami Naicker	35	M	N1 Ns
49	43	Dhanabagiam	35	F	N1 Ns
50	"	Alagamma	35	F	Tbd Minor
51	41	Kandiswamy	25	M	N1 Ns
52	39	Ammakannu	48	F	N1 Ns (patch on rt cheek)
53	"	Arunachalam	18	M	N1 Nt
54	30	Muthummal	50	F	N1 Ns
55	30	Kannappa Devan	55	M	L1
56	Hut	Iatnam	38	M	L1
57	Hut	Jayaram Naicker	50	M	L1
<i>Old Mambalam Road</i>					
58	11	Kristappa Chetty	58	M	N2 Ns
59	12	Doraiswami Naicker	30	M	N1 Ns
60	21	Lakshmi	45	F	N1 Nt
61	Hut	Muruswami Naidu	25	M	N1 Ns
62	34	Subbatammal	40	F	N1 Ns
63	Hut	Kuppan	25	M	N1 Ns
64	22	Kumaraswami Pillai	45	M	N1 Ns (suspicious)
65	Hut	Shanmugham	25	M	N1 Ns
66	51	Kannamma	55	F	N1 Ns
<i>Thoppet Street</i>					
67	45	Covinda Chetty	40	M	N1 Ns (patch)
68	44	Subramaniam	35	M	Tbd Minor rt ear lobe (suspicious)
69	50	S V Manikka Mudahar	28	M	L1
70	"	Valliammal	40	F	N1 Nt Minor
71	"	Rubavathi ammal	20	F	Suspicious
72	22	Annapoorna	16	F	L1
73	56	Kannappan	15	M	N1 Nt

ADULTS—continued

Serial No	Door No	Name	Age	Sex	Remarks
<i>Thoppet Street—continued</i>					
74	57	Dakshinamoorthy	17	M	L1
75	60	Appavoo Mudali	31	M	N1 Ns
76		Balakrishnan	18	M	Suspicious
77	62 63	Sambadan	17	M	Suspicious
78	64	Kamakshee	40	F	N1 Ns
79	75	Elumalai	20	M	N1 Ns
80	79	Kamalvee	44	F	N1 Ns
81	80	Appavoo Chetty	40	M	N1 Ns
82	2	Abalu Chetty	40	M	L1
83	1	Ethurajulu Naicker	56	M	N3
84		Ekambara Naicker	35	M	N1 Ns (suspicious)
85	9	Lakshmanan	20	M	N1 Ns
86		Munuswamy	30	M	N1 Ns
87	13	Dhanabaghyam	16	F	N1 Ns
88	14	Vedaguri	18	M	N1 Nt Major
89		Kuppu Chetty	35	M	N1 Ns
90	16	Vedachallam	22	M	L1
91	23	Srinivasan	30	M	N1 Nt Minor
92	24 25	Murugesu Mudahar	30	M	Patch on rt shoulder
93	28	Munuswamy Mudahar	30	M	Tbd Minor (suspicious)
94	33	Parvathy	50	F	N1 Ns
95	35	Natesan	25	M	Tbd Minor
96	37	Tholasi Naicker	35	M	L1
97		Jambulinga Chetty	26	M	N1 Ns
98	17 (back)	Arunachallam	50	M	N1 Ns
<i>Reddikuppam Road</i>					
99	2	Govindammal	35	F	N1 Ns
100	9	Pavadar	38	M	N1 Ns
101		Kannan alias Munuswamy	25	M	N1 Ns
102		Kanniah Naidu	30	M	Tbd Minor lt cheek
103		Arumugham	32	M	N1 Nt Minor
104		Palani	25	M	Open case
105		Dakshinamoorthy	22	M	N1 Ns
<i>Thoppet Street—continued</i>					
106	73	Manickammal	25	F	Suspicious
107		Kamakshee	17	F	N1 Ns
			M	F	
Definite cases			67	33	Total 100
Suspicious cases			6	1	Total 7
			73	34	107

SURVEY TYPE I and II

SCHEDULE I

District Chingleput

Taluk Sandapet

Village Area II

Total Population	Sex	Cases Detected			Incidence per 1000
		Neural	Lepromatous	Total	
1650	Males	101	17	118	60.4
1408	Females	64	9	73	
3158	Total	165	26	191	

ANALYSIS OF CASES BY TYPE AND AGE

Age Group	Cases				Total for Age Group	Percentage of Total Cases
	Neural		Lepromatous			
	No	Percentage	No	Percentage		
0-14	82	90.1	9	9.9	91	47.6
15-34	38	80.9	9	19.1	47	24.7
Over 34	45	84.9	8	15.1	53	27.7
Total	165	86.3	26	13.7	191	100.0

III REPORT OF LEPROSY SURVEY OF (KALAMPATU) MOTTU VILLAGE IN NORTH ARCOT DISTRICT

Situation of the Village

This is a small village consisting of a single street with a total population of 350. It is situated in a valley between two hills and is about five and a half miles from the village Latten within five miles of Katpadi a railway station on the main Madras Bangalore line. From Latten the approach to the village is by a crude country cart route which leads up to another village Kalampatu. From the last village one has to walk about three quarters of a mile through fields.

Main Occupation in the Village

Agriculture and illicit distillation of 'Arrack. The latter is largely for the villagers own use and clandestine sale to outlying villages

Climate

Dry area with a relative low temperature during nights

Water-supply

From wells most of which are used for irrigation purposes

Leprosy Conscience

The late forms of the disease are recognised but the early manifestations of the disease are ignored. There seems to be considerable knowledge of leprosy. The weekly ambulance of the Missionary Medical College Hospital stops at Latteri and as a result some propaganda has been done and patients treated

Diet

The staple diet is chiefly Pagi Cholam (local grains) occasionally vegetables buttermilk etc. Rice is not strangely enough the staple diet. The former grains are of fair nutritive value and better than rice

Prevalence of Skin Diseases

Dermatitis scabies and fungus infection of the skin are common

Leprosy Statistics

	Adults and Children			Adults			Children		
	Total	M	F	Total	M	F	Total	M	F
Population	325	172	153	234	124	110	91	48	43
Number examined	314	168	146	227	122	105	87	46	41
Number of leprosy cases	39	20	19	26	18	8	13	9	4

Percentage of population examined

Gross incidence

Sex incidence

Child incidence

Sex rate

Child rate

Open case rate

Suspicious cases

96.6%

124 per 1 000

14.9% males

11.6% females

14.94%

64.1%

33.3%

30.77% (this included open cases
(2) died within two years)

Total 3 (2 adults and 1 child)

DEFINITE CASES

	Ni Ns	Ni Nt	Leptomatous	N Incipient
Adults	11	6	9	Nil
Children	6	11	1	Nil

Interesting Features of the Survey

(a) The incidence of open cases in a small population of 325 is rather strikingly high

(b) The absence of serious lesions in children was an interesting feature. The tuberculoid case invariably showed a considerable degree of resolution. They were of the band like variety described by Dr Wade and others in a study of leprosy in Ceylon. Nerve enlargement was uncommon in one instance only was this present.

(c) The distribution of cases was mostly around limited foci of infection and where leptomatous cases were found to reside or have resided.

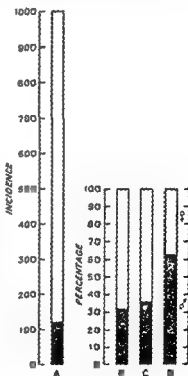
Leprosy History and Distribution of Cases in the Village

About twenty years back the only person who had leprosy in the village was said to be the father (dead) of Jengam residing in house No 25 (see map). The open case—Varayanaswami Chetty—in house No 30 developed it later. Peddappa, another open case is the son of Varayanaswami Chetty. The second focus was house No 1 where the husband of one Cajarammal (recently died) had leprosy. Cajarammal also developed leprosy, both are said to be open cases. The third focus is around house No 7 and 8 (refer map) where the husband of Boolokammal was said to have been an open case. The brothers of this man living opposite are all of them simple neural cases. Boolokammal herself is another open case. Her son Desvarajulu is an open case. Just opposite where the brothers in law of Boolokammal live another girl Simpoornam aged fourteen years is also an open case. Her father as stated earlier is a simple neural case. This whole family constitutes a group of cases. From Boolokammal infection seem to have spread to No 7 and 7a houses which are just at the back within easy approach. There are two other open cases in houses No 4 and 5 which are also easily accessible to houses No 7 and 8 as will be seen from the map. All other cases are around these three foci. The whole village is said to be interlarded

A GROSS INCIDENCE 124 PER 1000

B OPEN CASE RATE 30.77 /

C CHILD RATE 33.3 /

SEX RATIO $\frac{\text{MALES 149 PER 1000}}{\text{FEMALES 96 PER 1000}} = 1.6$ 

Practice of Isolation

Obviously bad cases seem to be isolated (food is given to them separately. For instance Nuriyanaaswami Chetty and his son Peddappa in house No 30 live in isolation in a separate hut at the back yard

Local Conditions Affecting Spread of Leprosy

It is said that this village is isolated by the neighbouring villages for fear of leprosy. Children from houses with leprosy are said to be admitted with difficulty into village schools. This has resulted in an attempt on the part of the parents to hide boys of school age going for examination. When found it was requested that the knowledge be kept secret. The opinion of the neighbouring villages is that Mottur is full of leprosy so much so that people fear to go into the village. Possibly this story is purposefully spread as a cover for the illicit distillation of arrack.

Indigenous Method of Treating Tuberculoid Leprosy

A leaf is ground and applied over the lesion in the form of a paste. It appears to be caustic in nature. Actually this was seen being applied on two of the children examined. The edges of the lesion showed evidence of irritation and ulceration. There were other cases of a similar nature.

SUPPLY TYPE I**SCHEDULE I***District* North Arcot*Taluk**Village* Mottur

Total Population	Cases Detected				Incidence per thousand
	Sex	Neural	Lepromatous	Total	
168	Males	17	9	26	124
146	Females	12	1	13	
314	Total	29	10	39	

ANALYSIS OF CASES BY TYPE AND AGE

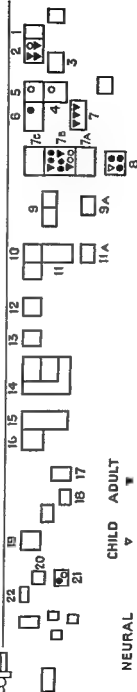
Age Group	Cases				Total for Age Group	Percentage of Total Cases
	Neural		Lepromatous			
	No	Percentage	No	Percentage		
0-14	12	92.3	1	7.7	13 (M 9 F 4)	33.3
15-34	4	50.0	4	50.0	8	20.6
Over 34	13	72.2	5	27.8	18	46.1
Total	29	74.4 ¹	10	25.6 ¹	39	100.0

¹ The percentages indicate the proportion of N and L cases.

PLAN OF (KALAMPTU) MOTTUR VILLAGE GUDIYATHAM TALUK N A DT



TEMPLE



CHILD ADULT

NEURAL
LEPROMATOUS

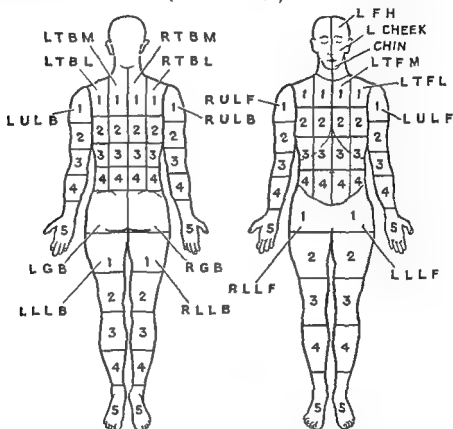
Serial No	Door No	Name	Sex	Age	Caste	Type of Disease and Remarks
1	1	Munirathnam	Mc	15	Chetty	1 N1 Ns (suspicious)
2		Dhanabai	Mc	13		1 N1 Nt (suspicious)
3	2	Govinda Naidu	M	37		L2
4		Gopal	Mc	12		N1 Incipient
5		Chakrapani	Mc	13		N1 Ns
6		Mohanraj alias Chunnappa	Mc	8		N1 Ns
7	4	Chinnaswamy	M	21		L1
8	5	Chillakandan	M	11		L1
9	6	Papiah Naidu	M	45		N2 Ns
10	7	Govindan	Mc	10		N1 Nt
11		Munirathnam	Mc	10		N1 Ns
12		Adilakshmi	F	7		N1 Nt
13		Ellammal	F	70		N1 Nt (not verified)
14	7B	Govindaraja	Mc	7		N2 Nt
15		Govinda Naidu	M	50		N2 Ns
16		Rajammal	F	37		N2 Ns
17		Sriramulu	Mc	16		N1 Ns
18		Hammal	F	8		N1 Nt
19		Chinnappa ammial	F	35		N1 Nt
20		Boolekammal	F	38		L1 (husband died open case)
21		Devarajan	M	18		L1
22		Kannan	Mc	6		N1 Nt Major
23	8B	Sumpoornam	F	14		L1
24		Babulu Naidu	M	43		N2 Ns
25		Srinivasan	M	33		N1 Ns
26		Kuppuswami Naidu	M	40		N1 Ns
27	18	Rajammal	F	23		N1 Nt (not verified)
28	21	Lakshmi	F	37		N1 Nt Major
29		Mayan	M	Adult		L1
30	24	Gangammal	F	43		N1 Nt Major
31	26	Ellammal	F	73		N1 Nt
32	27	Pattabi	M	Adult		N1 Nt (not verified)
33	28A	Chakrapani	Mc	7		N1 Ns
34		Sakunthala	F	11		N1 Nt
35	28B	Chinnappan	M	23		N1 Ns
36		Varadachari	M	20		1 N1 Ns (suspicious)
37	28C	Krishnan	M	19		N1 Ns
38	30	Narayanasami Chetty	M	58		L2 N3
39		Ieddappa	M	23		L2
40	31	Chellammal	F	41		N1 Ns
41		Muthulu	Mc	10		N1 Ns
42	33	Abboy Naidu	M	37		N1 Ns
43	34	Lakshmanammal	F	70		N1 Ns
44	35	Chinnammal	F	38		N1 Ns
45	39	Kanthammal	F	22		N1 Ns
46	40	Perruswamy	M	29		N1 Ns

REMARKS OF HON. DIRECTOR LEPROSY CAMPAIGN ON VERIFICATION OF CASES
IN THE VILLAGE

- No 1 and 2 taken as suspicious because of the absence of definite signs though strongly suggestive in view of the leprosy history in the family
- No 4 Studying at Vellore Absent from village Not verified not included in the statistics
- No 13 Not verified owing to absence from village Reported by Dr Sarma as a definite Nt Nt case (retrogressed) Not included in the statistics
- No 16 17 and 18 Absent from village but appear to be definite cases since they are taking treatment in the Vellore Mission Out-patient Hospital Car along with father of No 13 These are included in the statistics
- No 21 Not verified Absent Reported to be definitely Lt by Dr Sarma I am inclined to think it is a definite Lt since the mother is an open case And Dr Sarma has little doubt about the case
- No 27 Not verified Not included in the statistics
- No 9 Reported to have left for Chingleput Seeking admission Of Vellore Mission Lt case Included in the statistics
- No 37 Absent from village not verified not included in the statistics
- No 30 Typically suspicious patch but no definite signs hence included in the suspicious category
- No 46 Appears to be an Nt Nt case but it is reported that the case was examined at the Vellore Mission Hospital and smears found to be positive for bacilli hence taken as Lt

APPENDIX III

DIAGRAMMATIC METHOD OF RECORDING INTRADERMAL INJECTIONS ON THE BODY
 THAT THE PHYSICIAN CAN SEE AT A GLANCE THE EXACT POSITION OF THE PREVIOUS
 INTRADERMAL INJECTION (MUIR'S METHOD)



The front and back of the body is divided into four areas as follows

- LTBM
equivalent to left trunk back middle
- RTBM
equivalent to right trunk back middle
- LTFL
equivalent to left trunk front middle
- RTFL
equivalent to right trunk front middle
- LTBL
equivalent to left trunk back lateral
- RTBL
equivalent to right trunk back lateral

These areas are then numbered down the body 1 2 3 and 4

Similarly the arms and legs are divided into five areas as follows

L U L B

equivalent to left upper limb back

1 2 3 4 5

L U L F

equivalent to left upper limb front

1 2 3 4 5

R L L F

equivalent to right lower limb front

1 2 3 4 5

L L L F

equivalent to left lower limb front

1 2 3 4 5

Areas of the face are indicated as follows

L F H

equivalent to left forehead

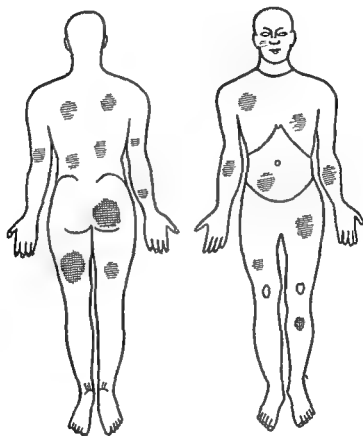
R F H

equivalent to right forehead

Right cheek right chin and so on

When a patient comes for his injection the physician marks with a slim pencil the place on the body to be injected and records this area according to the symbols described above

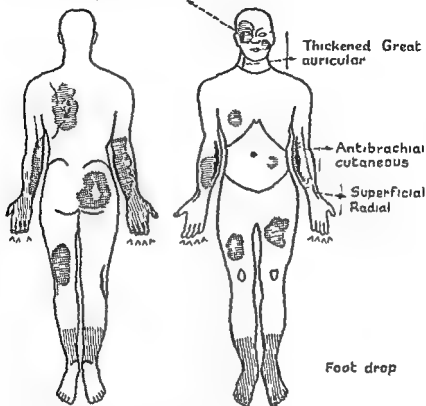
APPENDIX IV

1 DIAGRAMMATIC REPRESENTATION OF LESIONS IN SIMPLE MACULAR LEPROSY (NEURAL TYPE)— N_1^{NS} 

- Hypopigmented macule and definite margin
- Simple hypopigmented macule anaesthetic

2 DIAGRAMMATIC REPRESENTATION IN LESIONS OF NEURAL TUBERCULOID LEPROSY (MAJOP)—A₂VT

Lagophthalmos paralysis of superior orbicularis



Infiltrated erythematous raised anaesthetic circumscribed macule



Superficial cutaneous nerves going into macule



Ulcerated centre — Reactive phase



Apparently normal area in the centre of a major Tubercloid macule



Desquamating exfoliative or Scaly macule Reactive phase

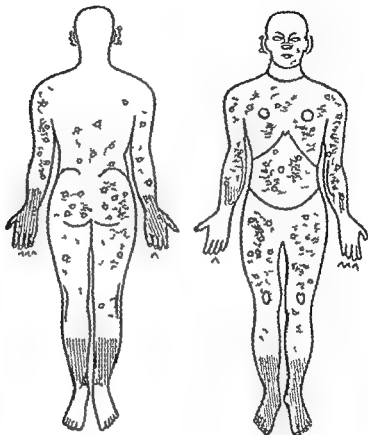


Nerve abscess



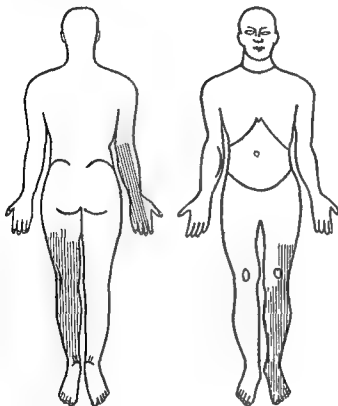
Saccular irregular nerve thickenings

3 DIAGNOSTIC REPRESENTATION OF ADVANCED LEPROMATOUS LEPROSY— L_3



- | Thick infiltration Ears
- Nodules Ears
- ◯ Depressed bridge of nose
- Gynecomastia
- Small Nodule
- ◯ Large sub-cutaneous nodule
- ^^ Contractures Fingers

4 DIAGRAMMATIC REPRESENTATION OF LESIONS OF
NEURAL ANAESTHETIC LEPROSY—N₁La



) Thickening of nerve (ulnar & peroneal)

||||| Anaesthesia

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